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EARLY DIAGNOSIS OF POLIOMYELITIS

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Treatment of poliomyelitis should begin as soon as possible after the onset of the disease in order to relieve the discomfort of patients and to minimize crippling. An analysis of 1,125 cases of poliomyelitis treated in Minneapolis during the 1946 epidemic reveals that the symptoms and observations are sufficiently characteristic to enable the diagnosis to be established in most patients within twenty-four hours of the onset. The study also discloses that paralysis is not a useful diagnostic sign because paralysis or weakness of the muscles is not a common early event and in a considerable number of cases does not occur at all. To await the appearance of paralysis to confirm the diagnosis or to begin treatment is inadvisable. It is now recognized, but should be emphasized, that patients without paralysis are often seriously ill and may become crippled from conditions other than paralysis which affect the muscles. All cases should be brought under treatment as soon as possible.

While it is known that the disease varies in the intensity of its attack the early medical symptoms are common to all types of cases including those which do not at any stage show evidence of weakness of the muscles. In this series of 1,125 cases 65.4 per cent (736 cases) progressed to paralysis of some degree but 34.6 per cent (389 cases) did not show paralysis at any time. The mortality was 6 per cent (68 patients), indicating that the epidemic was of average severity and that the observations drawn from the study of these cases are representative of the disease.

EARLY SYMPTOMS

Headache was the most distinctive initial complaint denoting the onset of poliomyelitis. This was usually of a severe, generalized, unrelenting type, which is rare in the illnesses of childhood. Headache was a particularly prominent symptom in 57 per cent of all the patients in this series during the first twenty-four hours of the disease.

Fever was noted early in the disease, but fever is not distinctive as a symptom. In this series fever was recorded in 80 per cent of the patients within the first twenty-four hours of the illness. The elevation of temperature in poliomyelitis is usually moderate and is seldom observed to rise above 103 F.

In addition to headache and fever the principal symptoms of the disease (in the order of frequency of occurrence during the first twenty-four hours) were nausea, vomiting, anorexia, stiff neck, stiff back, painful extremities and general malaise with listlessness.

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It has been stated frequently that the early symptoms of poliomyelitis may be indistinguishable from those of the more ordinary diseases affecting the nose, throat and respiratory system. Although 4 per cent of the patients in this series complained of head cold or respiratory infection attributed to the onset of the disease, the complaint was not generally confirmed on physical examination.

Sore throat or tonsillitis was an early complaint in 10 per cent of the patients, but inflammation was not found to be present in the physician's examination of these patients. The absence of inflammation of the throat in patients who complain of sore throat suggests the important possibility that the lesion is a "painful throat" of neuritic origin. The complaint of sore throat occurred ten times more frequently in the cases in which bulbar symptoms ultimately developed than in those which eventually showed paralysis of spinal origin. The inference to be drawn is that patients complaining of sore throat should be watched closely for evidence of bulbar paralysis.

A feature of poliomyelitis which may result in unnecessary delay unless kept in mind is the tendency in some cases for the symptoms to abate temporarily after an initial illness which usually lasts twenty-four hours. This type of history was obtained in 22 per cent of the cases in this series. Scrutiny of the histories of these patients reveals that the early symptoms were definite and recognizable. The significance of the recurrence of symptoms after a remission of two to three days was not apparent and did not seem related to the onset of paralysis. The interval or recurrent type of onset was present in 17 per cent of the patients in whom paralysis never developed. An opportunity was afforded to study 39 patients admitted to one hospital unit on the day of onset of the disease. None of these patients who were hospitalized early showed the phenomenon of recession and recurrence of symptoms, although in several paralysis developed after admission to the hospital.

PHYSICAL EXAMINATION

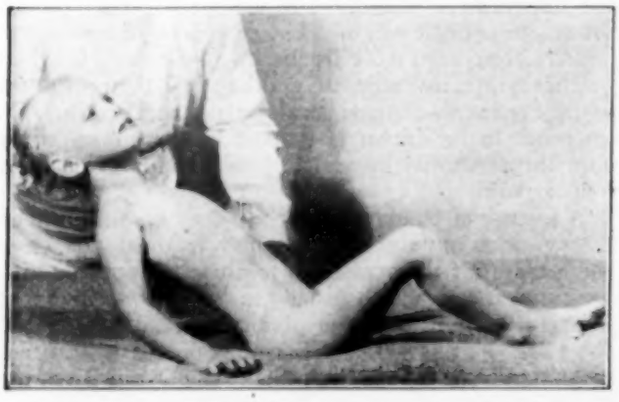
The diagnosis of poliomyelitis can be established with a high degree of certainty by physical examination. The typical patient lies quietly in bed and does not wish to be disturbed. There is a singular irritability and resentment against being moved or handled. The painful sensitivity of the peripheral tissues indicates the presence of spasm, a common and useful observation for the purpose of diagnosis early in the disease. Stiff neck, stiff back and shortened hamstrings are familiar examples of symptoms caused by the disease, but muscles elsewhere in the body may also be involved by spasm, including those which are not attached to the spine. The disorder occurs in the nonparalytic as well as the paralytic case.

For examination the patient is placed supine on a firm bed or table. On placing the hand beneath the head and attempting to flex the neck resistance will almost invariably be encountered as early as clinical symptoms have occurred. An attempt to flex the body will disclose stiffness of the back. When the trunk is flexed the knees will rise from the bed because of shortness of the hamstring muscles, as shown in the accompanying illustration. The stiffness is due to the condition of spasm in the posterior muscles. On search, the same hypertonicity and shortening may be found, although less frequently, in other muscles of the body and in all types of cases (table 1).

The patient may complain of pain in the muscles involved by spasm, or pain may be elicited by gently stretching the affected muscles.

When the condition affects the muscles of any part of the respiratory mechanism the breathing of the patient may be impaired. Spasm may affect the abdominal muscles and simulate the spasm of acute appendicitis.

It will be noted from table 2 that spasm occurs more frequently in the paralytic than in the nonparalytic type of case. Experience with this group of patients



Typical patient with poliomyelitis in the acute stage showing examination for spasm in the posterior muscles.

has shown that spasm is severer in the paralytic than in the nonparalytic case and is more difficult to relieve. In this series of cases (1,057 surviving) it was necessary to treat the nonparalytic patients (389) for an average of twenty-three days before the spasm was relieved, whereas from two to ten months (depending on its severity) was required to relieve the spasm in the paralytic cases (668).

PARALYSIS IN DIAGNOSIS

Paralysis or weakness of the muscles is easily discoverable when it occurs in poliomyelitis, but the value of this observation for the purpose of diagnosis is diminished by the fact that paralysis is not a common early condition and may not occur at all.

There was no detectable evidence of weakness or paralysis at any time in 389, or 34.6 per cent, of the 1,125 cases in this series. Paralysis occurred at some time within fifteen days of onset in 736, or 65.4 per cent, of the patients.

None of the patients complained of weakness or paralysis as an initial symptom, and this condition appeared during the first twenty-four hours in only 5 per cent of the cases (table 3). In 11.7 per cent of the patients the date of development of paralysis could not be determined from case records; but granting that these might have occurred within the first few days of illness, paralysis would still not be considered a common early symptom. It will be noted from table 3 that in 9.6 per cent of the patients paralysis did not occur until after the disease had been in progress for more than seven days.

TABLE 1.—*Frequency of Occurrence of Spasm on the Basis of Clinical Examination of the Total Surviving (1,057) Cases of Acute Poliomyelitis*

Muscle in Spasm	Occurrences	
	No. of Cases	% of Total Cases
Posterior neck	1,057	100.0
Posterior spinal	1,057	100.0
Hamstrings	1,057	100.0
Calf	846	80.0
Tensor fasciae femoris	560	53.0
Upper trapezius	220	20.8
Plantar fascia	153	14.5
Quadriceps femoris	103	9.7
Pectoralis major	98	9.3
Quadratus lumborum	70	6.6

The finger flexors, arm adductors, abdominals and biceps brachii were involved to the extent of 2 per cent of the cases and other muscles to a lesser extent.

Patients who manifested bulbar symptoms, such as difficulty in swallowing, nasal regurgitation, facial weakness, nasality and difficulty in speech, gave evidence of muscular dysfunction, on the average, slightly earlier than did patients in whom the spinal type of paralysis developed (table 4).

EXAMINATION OF THE SPINAL FLUID

The spinal fluid should be examined in every suspected case as an aid not only in confirming the diagnosis but in quickly separating out other diseases. The typical spinal fluid of poliomyelitis has the following characteristics:

Crystal clear.
Pressure normal or low.
Protein slightly elevated or normal.
Cell count moderately elevated, usually not above 350 per cubic millimeter. (Cell counts were not elevated in 4 per cent of the cases in this series in which other observations served as the basis of diagnosis. The cell count was normal in 1 per cent of the patients in whom paralysis occurred.)

TABLE 2.—*Occurrence of Spasm on the Basis of Clinical Examination of Paralytic and Nonparalytic Types of Acute Cases of Poliomyelitis (1,057 Total Living Cases)*

Muscle in Spasm	Occurrence of Spasm			
	668 Paralytic Cases		389 Nonparalytic Cases	
	No. of Cases	% of Cases	No. of Cases	% of Cases
Posterior neck	668	100.0	389	100.0
Posterior spinal	668	100.0	389	100.0
Hamstrings	668	100.0	389	100.0
Calf	556	83.4	290	74.6
Tensor fasciae femoris	374	56.0	186	47.9
Upper trapezius	173	25.9	47	12.1
Plantar fascia	124	18.6	29	7.5
Quadriceps femoris	93	13.9	10	2.6
Pectoralis major	95	14.2	3	0.8
Quadratus lumborum	55	8.2	15	3.9

The polymorphonuclear cells frequently predominate during the first forty-eight hours of the disease, after which time the majority of the cells are usually of the mononuclear variety.

Other of the usual laboratory studies of the spinal fluid or blood have not yet proved useful, as no changes have so far been disclosed which are particularly distinctive of poliomyelitis. Such examinations, however, are helpful in establishing a discriminative diagnosis in suspected cases which are under observation.

DIFFERENTIAL DIAGNOSIS

The diseases affecting the nervous system which are commonly confused with poliomyelitis are Guillain-Barré, encephalitis, meningitis and transverse myelitis. Guillain-Barré disease is noteworthy in contradistinction to poliomyelitis for the absence of stiffness of the neck and for the tendency of weakness or paralysis to progress over a period of weeks. The spinal fluid typically shows a high protein content without an increased cell count.

The patient with meningitis or encephalitis is usually restless and hyperactive at the onset in comparison with the listlessness and lethargy of the poliomyelitis patient. Transverse myelitis is marked by sensory changes, most unusual in poliomyelitis, in addition to the weakness.

TABLE 3.—*Occurrence of Paralysis in 1,125 Cases of Acute Poliomyelitis*

Day of Occurrence	No. of Cases	% of Cases
First	56	4.9
Second	82	7.3
Third	119	10.7
Fourth	89	7.9
Fifth	59	5.2
Sixth	52	4.6
Seventh	40	3.5
Eighth through fifteenth	108	9.6
Undetermined *	131	11.7
Total	736	65.4
Total paralytic cases	736	65.4
Total nonparalytic cases	389	34.6
Total cases	1,125	100.0

* The day of onset of paralysis was not known in 131 paralytic cases including the 68 deaths.

TABLE 4.—*Occurrence of Paralysis in Spinal and Bulbar Types of 605* Acute Cases of Poliomyelitis*

	First Appearance of Paralysis			
	431 Spinal Cases		174 Bulbar Cases	
Day of Occurrence	No. of Cases	% of Cases	No. of Cases	% of Cases
First	37	8.5	19	11.0
Second	56	13.0	26	15.0
Third	81	18.7	38	21.8
Fourth	61	14.3	28	16.1
Fifth	41	9.5	18	10.3
Sixth	38	8.8	14	8.0
Seventh	31	7.2	9	5.2
Eighth through fifteenth	86	20.0	22	12.6
Total	431	100.0	174	100.0

* The day of onset of paralysis was not known in 131 paralytic cases including the 68 deaths.

CONCLUSION

In the acute stage of poliomyelitis the principal manifestations in the peripheral structures which are of use in diagnosis are spasm (shortening of the muscles) and paralysis (or weakness of the muscles). Spasm appears early and is present in every case. Paralysis is easily discoverable when it occurs, but it is not a common early observation and does not appear at all in a considerable number of cases. Spasm is generally severer in the paralytic patient; the longer it is untreated the more difficult it is to relieve. In order to aid the prevention of contractures and deformities from spasm it is advisable to begin treatment to the peripheral tissues before weakness or paralysis has occurred. No delay in treatment is prudent once the diagnosis has been established.

OCULOGLANDULAR TULAREMIA TREATED WITH STREPTOMYCIN

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Lesions resembling oculoglandular tularemia were first noted by Martin [1] in 1907. He described 3 cases of conjunctivitis in patients with what he called "rabbit fever." It was not until 1911, however, that the causative organism was first isolated by McCoy and Chapin,[2] who were investigating a disease of ground squirrels in Tulare county, California. The organism was named Bacillus tularense after the county where it was first discovered. Fourteen years later one of Martin's patients was found to have a positive agglutination test for tularemia.[1] In 1913 Vail [3] saw an unusual form of conjunctivitis and Wherry and Lamb,[4] working on this case, were the first to isolate Bacterium tularense in pure culture from a human being.

At first it was thought that rabbits alone were responsible for transmitting this disease to man, but it has since been shown that many rodents, rabbit ticks, wood ticks, deer flies, coyotes, hogs, quail, pheasants and other animals and birds are hosts and transmitters of this infection to man.[5]

After the discovery of a causative organism tularemia began to be recognized in many parts of the United States, Canada and Europe. Francis reviewed 679 cases in 1928,[6] and he classified them in four main groups, as follows: (1) the ulceroglandular form, occurring most frequently and characterized by an open ulceration, regional lymphadenopathy and severe systemic manifestations out of proportion to the local lesion; (2) the glandular form characterized by regional lymphadenopathy in which a primary lesion is not evident; (3) the typhoid form in which both a primary lesion and lymphadenopathy are absent but in which constitutional symptoms predominate, and (4) the oculoglandular form in which the primary lesion is a conjunctivitis. In this series 32 cases, or 4.7 per cent, were of the oculoglandular form.

In 1939 Oosting [7] noted that there had been 68 cases of oculoglandular tularemia recorded up to that time, and he reported another. In 1940 Foshay [8] reported 600 cases of all forms of tularemia, of which 11, or 1.8 per cent, were of the oculoglandular form. In his series the primary lesion was found in the conjunctiva less often than in any other location. Francis [9] reported 78 cases in the United States and 18 in foreign countries up to 1940. Since then 8 cases have been reported in the

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2. McCoy, G. W., and Chapin, C. M.: Bacillus Tularense the Cause of the Plague-Like Disease of Rodents, Bulletin 53, United States Public Health and Maritime Hospital Service, 1912, p. 21.
3. Vail, D. T., Sr.: Bacillus Tularense Infection of the Eye, Ophth. Rec. **23**: 487 (Oct.) 1914.
4. Wherry, W. B., and Lamb, B. H.: Infection of Man with Bacterium Tularense, J. Infect. Dis. **15**: 333, 1914.
5. Simpson, W. M.: Tularemia: A Summary of Recent Researches with a Consideration of One Hundred and Three Dayton Cases, Ohio State M. J. **29**: 35 (Jan.) 1933.
6. Francis, E.: Symptoms, Diagnosis, and Pathology of Tularemia, J. A. M. A. **91**: 1155 (Oct. 20) 1928.
7. Oosting, M.: Oculoglandular Tularemia Contracted from the Tree Squirrel, Ohio State M. J. **35**: 730 (July) 1939.
8. Foshay, L.: Tularemia, Medicine **19**: 1 (Feb.) 1940.
9. Francis, E.: Oculoglandular Tularemia, Arch. Ophth. **28**: 711 (Oct.) 1942.

literature by Thompson and Glynn,[10] Gorrell,[11] Last,[12] Guerry[13] and Siniscal.[14] These and our own case bring to a total of 87 the recorded instances of oculoglandular tularemia in the United States. Undoubtedly many others have not been reported and many more have gone unrecognized. Nevertheless this small number serves to emphasize how unusual this manifestation of tularemia is.

SYMPTOMATOLOGY

Symptoms may come on quickly after an incubation period of two to five days. First the eyelids begin to swell, and then the preauricular, the submaxillary, the anterior cervical lymph glands and, sometimes, the axillary nodes become decidedly swollen and tender. As a rule, one eye alone is involved. On everting the eyelid, a pronounced degree of inflammation of the conjunctiva is seen, usually confined to the lower lid. Scattered through the red conjunctiva are small yellow nodules. These soon break down and leave shallow yellow ulcers, which stand out in well defined contrast to their scarlet background. The number of ulcers varies from one to ten, averaging about six. The cornea is involved only in the severer cases. With the appearance of the ocular lesions, constitutional symptoms become decided. Fever, chills, severe sweats and general malaise and weakness are the rule.

In a series reported by Vail in 1929[1] the ocular symptoms lasted about four weeks, the febrile reaction one to six months and general weakness of an incapacitating nature for six months or longer. The neighboring lymph glands remained large for many months after the primary lesion had disappeared. Francis[6] reviewed a large number of cases of all forms of tularemia and found that there was complete weakness for one month, weakness on moderate exertion during the second month and ability to do half time work during the third month of the disease. Many patients did not return to normal strength for six to twelve months. During convalescence there may be recurrent attacks of fever, sweats, attacks of extreme weakness and recurrent glandular enlargement. These recurrences may themselves last six to twelve weeks. All workers comment on the extraordinary variability of this disease, the source of infection, the modes of transmission and the clinical manifestations.

DIAGNOSIS

In his excellent review Francis[6] has emphasized four points that should lead to the diagnosis of tularemia: (1) a history of having dressed or dissected a wild rabbit, or of being bitten by a tick or a fly; (2) a primary lesion, such as a papule, a persistent ulcer or a primary conjunctivitis, followed by conjunctival ulceration; (3) persistent enlargement of the neighboring lymph glands, and, (4) fever of two or three weeks' duration.

The diagnosis may be confirmed by an agglutination test of the patient's serum. The reliability of this test is almost absolute. Its use makes diagnosis possible with a degree of accuracy seldom found in medical practice. Agglutinins are usually absent until some time during the second week. There follows an abrupt rise in titer during the third week, reaching a maximum from the fourth to the seventh week. The titer then gradually falls, and agglutinins may remain in low concentrations indefinitely. Human tularemia serums may also cross agglutinate Brucella abortus and Brucella melitensis organisms. In such cases the titer of the latter organisms will be less than the titer of Pasteurella tularensis. Isolation of the organisms from the conjunctiva or from the blood is difficult and requires specially enriched mediums and animal inoculation. For early diagnosis there is available a bacterial suspension intradermal test, which becomes positive during the first week.[15]

COMPLICATIONS

Complications are unusual in this disease. Those most likely to occur are ulcerative and suppurative adenitis, ulceration of the cornea and acute, purulent dacryocystitis. Francis has reported a case of ulceration with perforation of the ocular conjunctiva followed by prolapse of the iris.[9]

MORTALITY

In 1936 Pessin[16] reported the general mortality of tularemia to be 4.8 per cent in 5,426 cases. Francis[9] found a mortality of 6.9 per cent in 15,525 cases of all types of tularemia, with a mortality of 9 per cent in 78 cases of oculoglandular tularemia.

TREATMENT

Many methods of treatment have been tried. The most effective of all has been treatment with serum, developed by Foshay.[8] Even here, however, the high morbidity rate leaves much to be desired. The sulfonamide compounds, arsenic compounds, iodides, metaphen, acriflavine and penicillin have all been used without benefit.[17] Zeiter[18] and Siniscal[14] reported comparatively favorable results with the use of fever therapy on a few patients. Jackson[19] has reported favorably on the use of a solution of bismuth tartrate intravenously.

In 1944 Heilman's[20] experiments showed that the antibiotic substance streptomycin possessed considerable activity against Past. tularensis, as measured by in vitro and in vivo experiments. Lethal infections were established in mice, all of whom recovered when treated with streptomycin. Control animals failed to recover from the disease.

These experiments led to the use of streptomycin in the treatment of tularemia in human beings. Foshay and Pasternack[21] described 7 cases with early favorable results in all. None of these patients, however, had the oculoglandular form of tularemia. The experiences of Foshay and Pasternack compared favorably

10. Thompson, H. E., and Glynn, J. D.: Oculoglandular Tularemia, J. Iowa M. Soc. **30**: 70 (Feb.) 1940.
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16. Pessin, W. B.: Tularemia: Etiology, Diagnosis and Treatment, Wisconsin M. J. **25**: 448 (June) 1936.
17. Bell, J. F., and Kahn, O. B.: Efficacy of Certain Drugs in the Treatment of Tularemia, Arch. Int. Med. **75**: 155 (March) 1945. Josey, A. I.: Penicillin Treatment of a Case of Tularemia Without Effect, J. A. M. A. **126**: 496 (Oct. 21) 1944. Pullen, R. L., and Stuart, B. M.: Tularemia: Analysis of Two Hundred and Twenty-Five Cases, ibid. **129**: 145 (Oct. 13) 1945.
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19. Jackson, W. W.: The Treatment of Tularemia with Intravenous Bismuth Tartrate, Am. J. M. Sc., **209**: 513 (April) 1945.
20. Heilman, F. R.: Streptomycin in the Treatment of Experimental Tularemia, Proc. Staff Meet., Mayo Clin. **19**: 553 (Nov. 29) 1944.
21. Foshay, L., and Pasternack, A. B.: Streptomycin Treatment of Tularemia, J. A. M. A. **130**: 393 (Feb. 16) 1946.

with previous results when serum therapy was used. Abel [22] has also reported on the effectiveness of streptomycin in 3 cases.

The case of the oculoglandular type of tularemia reported here is of interest both because of its comparative rarity and because there has been no report up to the time of writing of the treatment of this disease with streptomycin.

REPORT OF A CASE

History.—A white boy aged 11 was hospitalized June 8, 1946, with swelling of his right eye of eight days' duration. Nine days previously his right eye had been struck by a stone. There was apparently no injury, as the incident was forgotten and he went in swimming later that day. The next day he complained of something in his eye, and the lids became swollen and reddened. His mother used eye washes of boric acid, and all discomfort disappeared. Seven days before admission, fever was noted. This persisted daily and was worse in the late afternoons than in the mornings. There were no chills, but there were severe sweats. The boy was kept in bed and except for mild generalized weakness and malaise had no complaints. The swelling about his eye gradually increased during this time. Family and past histories were noncontributory.

Physical Examination.—On admission the patient appeared to be a well developed and well nourished white boy, of good color and not in acute distress. The temperature by rectum was 104 F., the pulse rate was 100 and the respiratory rate 18. There was decided swelling and tenderness of the right eye. Both lids were edematous, and the palpebral conjunctivas were red and inflamed. On the lower right palpebral conjunctiva were scattered six small shallow irregular ulcers with yellowish bases. The bulbar conjunctiva appeared normal. The pupils were round, regular and equal and reacted to light and accommodation. The left eye appeared normal.

Examination of the ears, nose and throat revealed nothing except a mild pharyngitis. The chest was normal in appearance, size and shape. Resonance and breath sounds were normal, and adventitious sounds were not noted. Physical examination of the heart revealed no abnormality. The abdominal wall was soft, and there was no tenderness or rigidity. The spleen and liver were not palpated, nor were any other abdominal organs or masses palpable. The nervous system was normal. Skin and tendon reflexes were present and equal, and there was no evidence of meningeal irritation. The cranial nerves were normal.

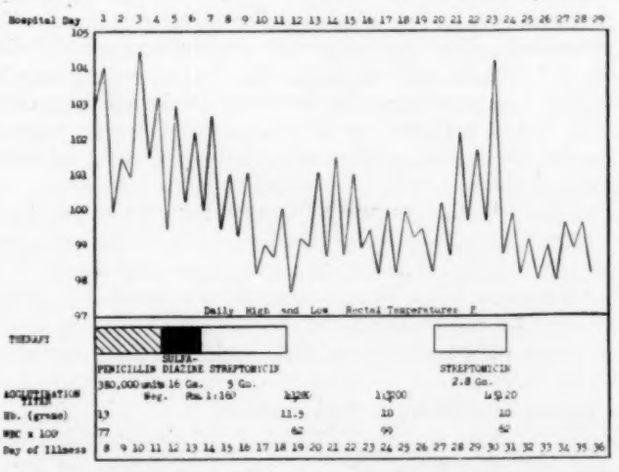
The right preauricular and submaxillary lymph glands were grossly enlarged and tender. All the lymph glands of the right anterior cervical group were likewise abnormally enlarged. Two large nodes were palpated in the right axilla. Throughout the rest of the body the lymph glands were normal in size and consistency.

Laboratory Examination.—Examination of the blood revealed a white cell count of 7,700, with 79 per cent polymorphonuclear leukocytes, 19 per cent lymphocytes and 2 per cent monocytes. The red cell count was 4,250,000, with a hemoglobin level of 13 Gm. per hundred cubic centimeters (Haden). Urinalysis, Kahn and Kline tests, and tuberculin tests were negative. Blood culture and culture from material on the palpebral conjunctiva were negative. These were planted only on routine mediums.

Course.—Penicillin therapy, 20,000 units at three hour intervals intramuscularly, was commenced. After three days the eye appeared to be worse. New conjunctival ulcers were forming, and the ocular conjunctiva was becoming involved. Further inquiry revealed that sixteen days before admission, while he was playing in the woods with another boy, our patient had found a dead rabbit. He handled the rabbit and carried it for a short distance. Later he was a close and interested bystander while his older playmate proceeded to dissect the animal. His playmate was wearing gloves at the time. The incident had already been forgotten and, of course, had not been considered significant by the parents.

With this knowledge, serum agglutination tests for tularemia, undulant fever and typhoid were commenced, and penicillin therapy was discontinued. Sulfadiazine therapy was initiated. The following day all agglutination tests were negative, but it was nevertheless felt that our patient did present the clinical picture of oculoglandular tularemia. A request for streptomycin was made to Dr. Chester S. Keefer, who generously arranged to have a supply sent to us. Before the streptomycin arrived no appreciable effect of therapy with sulfadiazine was noted. Streptomycin therapy was started late on the sixth hospital day and the drug was administered as directed, 1 Gm. daily dissolved in isotonic sodium chloride solution and given in divided doses every three hours intramuscularly for five days (1,000,000 units daily divided into eight doses of 125,000 units each). We were gratified to find that an agglutination test for tularemia was now positive in a dilution of 1 : 160, confirming our diagnosis.

The clinical response to therapy with streptomycin was striking. After twenty-four hours the edema was less and all tenderness had disappeared from the eye and regional lymph glands. As seen in the accompanying chart, the temperature



Clinical course of boy aged 11, weighing 32.7 Kg., admitted to hospital June 8, 1946 with oculoglandular tularemia and discharged July 6, 1946.

began to fall after twenty-four hours, reaching a normal level on the fourth day of therapy. After forty-eight hours the ocular conjunctiva had considerably improved, and swelling about the face and regional lymph glands was much reduced. Improvement proceeded so that after five days of therapy the only remaining evidence of disease was a mild palpebral conjunctivitis and small palpable lymph glands in the anterior cervical, axillary and preauricular groups.

A serum agglutination test for tularemia at this time was positive in a 1 : 1,280 dilution. The agglutination for Br. abortus was positive in a 1 : 40 dilution. Throughout the treatment period the patient was observed for possible toxic effects of streptomycin, but untoward effects were not noted.

After streptomycin therapy was discontinued there began again a gradual daily rise in temperature to levels of 100 and 101 F. The general condition of the patient and the local appearance of the conjunctiva remained good and except for fever there was no sign of illness or disability. In order to forestall a long-drawn-out convalescence, a second lot of streptomycin was generously sent to us from another company, and treatment was started again on the twentieth day. The dosage and method of administration was the same as with the first course. This time, however, there was much pain and tenderness at the sites of injection, and on the twenty-first day the temperature rose to 102.4 F. This continued until the twenty-second and

22. Abel, O., Jr.: The Use of Streptomycin in Tularemia, J. Missouri M. A. 43: 167 (March) 1946.

twenty-third days, when the temperature was 104 F. The administration of streptomycin was discontinued, since there was no other apparent cause for this fever, and the temperature immediately returned to normal and remained so for six days, after which our patient was discharged from the hospital. No other untoward effects that could be attributed to the drug were noted. At this time physical examination revealed an eye that was normal in all respects and a boy whose color was good and who was full of pep and energy, revealing no evidence of weakness or tiredness.

During the last few weeks in the hospital a gradually rising agglutination titer was observed. On the seventeenth hospital day, it was 1:3,200 for Past. tularensis and 1:80 for Br. abortus. On the twenty-fourth day it was 1:5,120 for Past. tularensis and 1:80 for Br. abortus. Shortly before discharge the hemoglobin content of his blood had fallen to 10 Gm. per hundred cubic centimeters, and the red blood cell count was 3,530,000. Examination of the blood revealed otherwise normal conditions.

Following discharge the boy was observed again at intervals during the next three months. His eye and his general condition remained normal during this period.

COMMENT

The first course of streptomycin produced gratifying results. This was similar to the observations of others.[23] The early relapse in our patient was not accompanied by the usual constitutional symptoms. He appeared and felt perfectly well during the entire febrile period. There was, in fact, difficulty in keeping him in bed and in persuading his parents that he was not well. The sharp sudden rise in temperature which accompanied the second course of treatment and its immediate subsidence after the drug was discontinued can evidently be attributed to the streptomycin rather than to the disease process. Either the second lot of streptomycin received was not as pure a product as the first or the patient had by this time become sensitized to the drug. The latter seems likely, since an apparent sensitization to streptomycin with violent febrile reactions during a second course of therapy has also been observed by Hinshaw and Feldman.[24]

There was no question in our minds, however, that the first course of therapy was responsible for rapid improvement in the primary lesion, the fever and the constitutional symptoms. The absence of fever during the patient's convalescence must also be attributed to the effect of streptomycin therapy.

In evaluating the effects of serum therapy, Foshay found that in 518 untreated cases, the average duration of disease was 3.8 months, the average disability was 3.36 months, fever 31.59 days and duration of the primary lesion 39.48 days. In our patient the duration of disease was 30 days, fever 30 days and the primary lesion 17 days. These figures compare favorably with those for untreated cases, as well as with Foshay's figures for 600 patients who received specific serum therapy.[8] On this basis at least it appears that streptomycin did favorably alter the expected course of the disease in our patient.

It is quite apparent that streptomycin has been in use for too short a period to properly evaluate its effectiveness in the treatment of human tularemia. Every indication at the present time suggests that it is a decided improvement over other forms of therapy. Future investigation may alter present methods of administration and dosage and improve further the therapeutic effect of the drug. It is certainly desirable to observe the results of more intensive and prolonged therapy in large numbers of patients before forming definite conclusions as to what results may ultimately be expected in the treatment of a disease that is naturally so variable.

SUMMARY AND CONCLUSIONS

A case of oculoglandular tularemia is presented together with a brief review of the literature. This is an unusual manifestation of tularemia and probably the first to be treated with streptomycin. There was immediate and dramatic improvement of the primary lesion, constitutional symptoms and fever, but the general course of the disease, as manifested by a return of low grade fever, was prolonged for thirty days. During convalescence a second course of streptomycin brought about symptoms which probably represented either sensitivity of our patient to streptomycin or an impurity in the drug. The course of the disease was shorter in all respects than that of the average untreated case.

334 North Oakhurst Drive.

23. Foshay and Pasternack.[21] Abel.[22]
24. Hinshaw, H. C., and Feldman, W. H.: Streptomycin in Treatment of Clinical Tuberculosis: A Preliminary Report, Proc. Staff Meet., Mayo Clin. **20**: 313 (Sept. 5) 1945.

IMMUNIZATION PROCEDURES IN PEDIATRICS

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It is the purpose of this paper to recommend certain preparations for active immunization against smallpox, diphtheria, tetanus and pertussis. These procedures have universal application to the practice of pediatrics. Active immunization against typhoid fever and scarlet fever will not be discussed in as much as it is necessary or advisable only within certain geographic limits. The recommendations made here are based on a review of the literature [1] biased, no doubt, by my own experience in pediatric immunity clinics. I will also briefly summarize results of a study of combined immunization with a mixture of diphtheria and tetanus toxoid containing Hemophilus pertussis vaccine.

SMALLPOX

Calf lymph virus has yet to be excelled as the preparation of choice for vaccination against smallpox. Failures are due to nonpotent lymph. The preservation of the vaccine at temperatures below 50 F. at all times and preferably in an electric refrigerator is essential to the maintenance of potency. Vaccination should always be on the arm or between the shoulder blades. Secondary infections are more likely to occur on the leg. The multiple pressure method should be used. A light sterile gauze dressing loosely applied is desirable to prevent fingernails from contact with the area. Vaccination may be performed at any time during the first year of life. During the first four months, however, occasional refractory persons are met with. Eczema is a positive contraindication. Revaccination should be done when the child starts to school.

From the Department of Pediatrics, Stanford University School of Medicine.

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1. Report of the Committee on Therapeutic Procedures for Acute Infectious Diseases and on Biologicals of the American Academy of Pediatrics, Evanston, Ill., 1945. The Control of Communicable Diseases, ed. 6, American Public Health Association, New York, 1945. Toomey, J. A.: Active Immunity, J. A. M. A. **119**: 18 (May 2) 1942.

DIPHTHERIA

Fortunately in this country there have not been any epidemics of malignant diphtheria. This being so the various types of diphtheria toxoid used in this country have not been subjected to the crucial test. In judging the products available one can only rely on tests of immunity.

Immunity in diphtheria is primarily antitoxic. It is the sum of existing circulating antitoxin and the capacity to manufacture more antitoxin rapidly on the slightest secondary stimulus. Existing circulating antitoxin can be measured by neutralization tests in laboratory animals. Capacity to react can be measured by a second determination after a reinjection. The Schick test not only measures the amount of antitoxin available at the site of injection but also the reactive capacity. For example, in previously immunized persons the injected Schick toxin may stimulate antitoxin rapidly enough to partially neutralize itself before injury occurs to the tissues and erythema results.[2] A Schick test thus serves incidentally as a small booster injection. Even though reports of malignant diphtheria in Schick-negative persons are now coming in, they are the exception rather than the rule. Comparison of antigens by means of the Schick conversion rate is valid. Furthermore, in the individual case in private practice the Schick test enables one to pick up the occasional poor reactor, provided that the test is performed more than three months after immunization.

Basic immunization should be performed during the first two years of life and, to be conservative, probably not before the eighth month, though recent evidence suggests that younger infants can be immunized. There is incontrovertible evidence, provided by the classic studies of Volk and Bunney,[3] that two injections of alum precipitated diphtheria toxoid provide a basic immunity slightly higher in degree and of longer duration than do three injections of fluid toxoid. The argument against alum precipitated toxoids, namely, the occasional formation of sterile abscess, has been practically nullified by improvement in the product. The injections should be given deeply and, in my opinion, intramuscularly, as Bousfield advises.[4] The interval between the injections should be one to three months. In the presence of an epidemic, this may be shortened to three weeks.

Fluid toxoid, three injections at monthly intervals, is second best and, as such, is not the agent of choice, at least in primary immunization. Fluid toxoid, however, is preferable in older age groups.[5] It should be used in exceedingly small doses, or suitably diluted, in those children who are sensitive to diphtheria bacillary protein. Sensitivity is determined by the intradermal injection of a 1:100 dilution of fluid toxoid. This test, known as the Moloney test,[6] is essential before primary or secondary immunization in persons 10 years of age or more. (I hope Dr. Thelander will discuss immunization in the older age groups from her large experience.)

2. Phair, J. J.: Diphtheria Immunization: The Interpretation of the Schick Test, Am. J. Hyg. **36**: 283, 1942.
3. Volk, V., and Bunney, W. E.: Diphtheria Immunization with Fluid Toxoid and Alum Precipitated Toxoid, Am. J. Pub. Health **32**: 690, 1942.
4. Bousfield, G.: Diphtheria Alum Precipitated Toxoid, Brit. M. J. **2**: 706 (Dec. 4) 1943.
5. Fraser, D. T.: Diphtheria Toxoid, Canad. Pub. Health J. **30**: 469, 1939. Smith, C. E.: Personal communication to the author.
6. Moloney, P. J.; Fraser, D. T., and Fraser, C. J.: Immunization Against Diphtheria with Toxoid, Ann. Inst. Pasteur **43**: 124, 1929.

There remains to be mentioned aluminum hydroxide adsorbed diphtheria toxoid. This has been used for years abroad.[7] In the remote community of Iceland, where only 10 per cent of the children under 13 years of age were naturally Schick negative, Sigurjonsson obtained 96 per cent Schick negative reactions seven months after two injections of this product at a three week interval. Four years later 74 per cent were still Schick negative. This is rather remarkable. In my own experience with aluminum hydroxide adsorbed toxoid, during the past four years at Stanford, I have yet to observe an injected child with a positive Schick test.

A fourth type of diphtheria toxoid, protamine-precipitated toxoid, has been reported recently. Antitoxin titers in a large group of children were unusually high.[8]

In conclusion, then, for basic immunity the conservative pediatrician should stand by alum precipitated toxoid. The more experimentally minded may with justification use the aluminum hydroxide adsorbed product.

When the child is 5 or 6 years of age and ready for kindergarten or school, should one perform a Schick test or simply give a booster injection? The public health physicians are warranted in omitting the Schick test and giving one reimmunizing injection.[9] In private practice, however, it is my belief that one should perform a Schick test, controlling it with a 1:100 dilution of toxoid.[10] If the test is negative, one need do no more. The toxin, and especially the diluted toxoid have served as little boosters. If the test is positive, two reinjections of alum precipitated toxoid should be given. The diluted toxoid control will have identified the few persons sensitive to diphtheria protein and thus enable the physician to proceed cautiously with reinjections.

TETANUS

Immunization against tetanus is quite a different problem from that against diphtheria. There are no naturally immune persons. Tetanus antitoxin has never been detected in nonimmunized men, except in China,[11] nor has it been found even after recovery from clinical tetanus.[12] Although tetanus spores are ubiquitous, they are of course not antigenic and, unlike the diphtheria bacilli in the street cars, they cannot restimulate the immunity mechanism. Only the toxin produced by the bacillary forms under anaerobic conditions is antigenic and then but feebly so.[13] Boyd [14] has attributed the 22 cases of tetanus which occurred in the immunized British forces to an inadequate level of circulating antitoxin.

7. Leach, C. N.; Jensen, C., and Poch, G.: Diphtheria Immunization with a Single Injection of Purified Formol-Toxoid and Aluminum Hydroxide, J. Lab. & Clin. Med. **20**: 451, 1935. Sigurjonsson, J.: Report on the First Protective Immunizations Against Diphtheria in Reykjavik, Deutsche med. Wchnschr. **62**: 1301, 1936; The Duration of Diphtheria Immunity, Ztschr. f. Hyg. u. Infektionskr. **122**: 189, 1940.
8. Ross, V.; Clapp, F. L., and Schimpf, B. W.: Antitoxin Titers Following Immunization with Protamine-Precipitated Diphtheria Toxoid, Am. J. Pub. Health **36**: 645, 1946.
9. Footnotes 3 and 5.
10. Brandon, K. F., and Fraser, D. T.: Observations on the Interpretations of Schick Tests, J. Immunol. **31**: 387, 1936.
11. Tenbroeck, C., and Bauer, J. H.: Studies on the Relation of Tetanus Bacilli in the Digestive Tract to Tetanus Antitoxin in the Blood, J. Exper. Med. **37**: 479, 1923.
12. Cowles, P. B.: Tetanus Immunization, Yale J. Biol. & Med. **9**: 409, 1937. Cooke, J. V., and Jones, F. G.: The Duration of Passive Tetanus Immunity, J. A. M. A. **121**: 1201 (April 10) 1943.
13. Zuger, B.; Greenwald, C. K., and Gerber, H.: Tetanus Immunization: Effectiveness of the Stimulating Dose of Toxoid Under Conditions of Infection, J. Immunol. **44**: 309, 1942.
14. Boyd, J. S. K.: Tetanus in the African and European Theatres of War, 1939-45, Lancet **1**: 113, 1946.

The aim in tetanus immunization, then, is to attain and maintain the highest degree of immunity at all times. After studying the initial lesions in 56 cases of clinical tetanus in children, Pratt[15] concluded that reduction in the incidence of this disease can only be accomplished if this aim is kept in view. The majority of his cases resulted from trauma or infection so trivial that they were overlooked either by the child himself, his parents or his physician. It is not plausible to expect parents to bring children into the office for a booster injection after each scratch. Antitoxic immunity, of itself protective without reliance on restimulation, is therefore the goal.

A great deal of evidence[16] has accumulated to indicate that alum precipitated tetanus toxoid produces a more durable immunity than plain or fluid toxoid. Two injections are usually advised, but three injections[17] are preferable. The interval should be three months. The first two injections may be combined with diphtheria toxoid. With this dosage the great majority of children will produce and maintain more than 0.1 unit of tetanus antitoxin per cubic centimeter of serum for one year. One tenth of a unit of tetanus antitoxin is believed to be protective of itself against such toxin as may be produced in trivial wounds.

Aluminum hydroxide adsorbed tetanus toxoid, alone or in combination with other antigens, may in the future equal or surpass the alum precipitated product as the agent of choice. Local reactions are less frequently encountered. Antitoxin levels produced are high and remarkably well maintained.[18]

A booster injection of 0.5 cc. alum precipitated tetanus toxoid should always be given one year after completion of the basic course. Thereafter reinjections should be repeated every two years. Annual reinjections with 0.25 cc. are more easily remembered. They can be given regularly at the time of the child's annual physical examination.

This schedule for immunization of course will be interrupted if the child is brought in to the office with an abrasion or anything more serious. A booster injection should then be given immediately. As 1 cc. fluid toxoid will raise the child's antitoxin titer more promptly than alum precipitated toxoid,[19] it is here the agent of choice. The minimum recorded incubation period of tetanus is as short as two days,[14] but incubation periods of less than seven days are rare. A booster injection cannot be expected to be of any value against such fulminating intoxication. The level of circulating antitoxin resulting from the child's previous injections of toxoid will be decisive. This level, however, can be temporarily increased by administering tetanus antitoxin (horse serum). In fact, resort to this old procedure is indicated, by no means contraindicated, in cases with extensive contaminated wounds. The recall injection of toxoid is injected simultaneously into another extremity.

15. Pratt, E. L.: Clinical Tetanus, J. A. M. A. **129:** 1243 (Dec. 29) 1945.

16. Bergey, D. H.: Active Immunization Against Tetanus Infection with Tetanus Toxoid, J. Infect. Dis. **55:** 72, 1934. Jones, F. G., and Moss, J. M.: The Antitoxic Titer of Human Subjects Following Immunization with Tetanus Toxoid and Tetanus Alum Precipitated Toxoid, J. Immunol. **30:** 115, 1936. Jordan, E. P., and Halperin, G.: Tetanus Toxoid for Prophylaxis, War Med. **1:** 227 (March) 1941. Newhouser, L. R.: Tetanus Toxoid Immunization, Mil. Surgeon **88:** 371, 1941. Miller, J. J., and Saito, T. M.: Concurrent Immunization Against Tetanus, Diphtheria and Pertussis, J. Pediat. **21:** 31, 1942.

17. (*a*) Bigler, J. A., and Werner, M.: Active Immunization Against Tetanus and Diphtheria in Infants and Children, J. A. M. A. **116:** 2355 (May 24) 1941. (*b*) Ygazell, L. A., and Deamer, W. C.: Response to a Stimulating Injection of Tetanus Toxoid, Am. J. Dis. Child, **66:** 132 (Aug.) 1943. (*c*) Miller, J. J., and Humber, J. B.: Observations on Tetanus Immunization: The Dosage of Alum Precipitated Toxoid and the Use of Fluid Toxoid After Trauma, J. Pediat. **23:** 516, 1943

18. Miller, J. J.; Humber, J. B., and Dowrie, J. O.: Immunization with Combined Diphtheria and Tetanus Toxoids (Aluminum Hydroxide Adsorbed) Containing H. Pertussis Vaccine, J. Pediat. **24:** 281, 1944.

19. Evans, D. G.: Active Immunization Against Tetanus, Lancet **2:** 628, 1941. Miller and Humber.[17c]

PERTUSSIS

The effectiveness of active immunization against pertussis has been proved by numerous studies in the field.[20] Occasional unfavorable reports can be accounted for by the use of nonpotent lots of vaccine. There is yet no United States standard of potency because there is no officially accepted means of assaying potency. Soon this may be remedied. Until then occasional nonpotent lots of vaccine may continue to appear from the best available sources. However, it must be admitted at once that in pertussis vaccine one does not have as almost universally potent an antigen as is available in either diphtheria or tetanus toxoid. Speaking conservatively, the total mass of evidence from field studies in different parts of the country indicates that H. pertussis vaccine will completely protect 2 of 3 children exposed in the family and 4 of 5 children exposed less intensely outside the home. Those vaccinated children who do contract pertussis usually have milder attacks and rarely die. Some apparent failures of immunization in vaccinated children are due to infections with the Bacillus parapertussis.[21] These infections will seldom be diagnosed correctly, as the cough is clinically indistinguishable from that of pertussis: in other words they are cases of whooping cough but not of pertussis.

The protective value of H. pertussis vaccines was proved in the field with simple saline suspensions of bacilli in the antigenic phase 1. All freshly isolated strains are in phase 1. Unless special precautions are taken, stock cultures will change into relatively nonantigenic phases. When saline suspensions are used the total number of bacterial cells injected should be at least 70 billion. Ninety to 120 billion cells are better. This total dose is divided into three injections and given preferably at three or four week intervals.[22] One week intervals, however, should be used during an epidemic. Suspensions containing 15 and 20 billion cells per cubic centimeter are, in my experience, less likely to provoke local reactions than more concentrated suspensions.

Again, as with the toxoids, an alum precipitated product is to be considered. Field studies and serologic evidence[23] indicate that 30 or 40 billion H. pertussis in the presence of alum are as effective as 80 to 100 billion bacterial cells in saline solution. Three injections of a fraction of a cubic centimeter each are given at monthly intervals. In my opinion, however,

20. Felton, H. M., and Willard, C. Y.: Current Status of Prophylaxis by Hemophilus Pertussis Vaccine, J. A. M. A. **126:** 294 (Sept. 30) 1944.

21. Eldering, G., and Kendrick, P.: Bacillus Para-Pertussis, J. Bact. **35:** 561, 1938. Bradford, W. L., and Slavin, B.: An Organism Resembling Hemophilus Pertussis, Am. J. Pub. Health **27:** 1277, 1937. Miller, J. J.; Saito, T. M., and Silverberg, R. J.: Parapertussis, J. Pediat. **19:** 229, 1941.

22. (*a*) Sauer, L. W.: Whooping Cough, in Brennemann, J.: Practice of Pediatrics, edited by I. MacQuarrie, Hagerstown, Md., W. F. Prior Company, 1945, vol. 2, chap. 34. (*b*) Lapin, J. H.: Whooping Cough, Springfield, Ill., Charles C Thomas, 1943.

23. (*a*) Daughtry-Denmark, L.: Whooping Cough Vaccine, Am. J. Dis. Child. **63:** 453 (March) 1942. (*b*) Kendrick, P. L.: A Field Study of Alum-Precipitated Combined Pertussis Vaccine and Diphtheria Toxoid for Active Immunization, Am. J. Hyg. **38:** 193, 1943. (*c*) Sauer.[22a]

the superiority of the alum precipitated vaccine has not been demonstrated in the age groups customarily immunized—that is, in infants aged 5 months or older. Unless combined immunization with alum precipitated toxoids, that is, a mixture, is to be used, I prefer to give the vaccine in saline solution in order to minimize local reactions.

This brings up the question of how early in life an infant can be actively immunized. Using an alum precipitated vaccine, Sako and his co-workers[24] in New Orleans conclusively demonstrated that injections begun between 1 and 2 months of age and repeated twice at monthly intervals (total dose 40 billion organisms), protected 81 per cent of infants against familial exposure. More important, none of their immunized infants who were attacked died. This well controlled field study was extremely important in that it opened the way for trials of active immunization during earliest infancy. Now, not only immunization against pertussis but against typhoid, diphtheria and tetanus is being attempted at an age at which it was formerly believed that the immunity mechanism was undeveloped. The evidence available[25] suggests that plain vaccine may possibly be less effective than alum precipitated vaccine in infants under 3 months of age.

There are on the market soluble antigens and endotoxoids of H. pertussis. The available evidence[26] indicates that they are of little or no prophylactic value. Only suspensions of whole cells in saline solution or with some adsorbent like alum should be used.

Complete protection against pertussis of ten years' duration may result in some persons from the primary course of immunization. Reinjections of vaccine, however, should be the rule. Whenever the child is known to have been intimately exposed a stimulating dose of 20 billion cells should be given.[27] Otherwise, in my experience, two reinjections with this dose (20 billion), the first about a year after the basic course and the second at the age of 6 years, are sufficient.

MIXED TOXOIDS AND VACCINES

It is apparent that if it is necessary to employ all the immunizing procedures to which I have alluded, the children's arms will resemble pin cushions. Mixtures of antigens will save the physician's time and the child's extremities, not to mention his tears. Ramon[28] challenged the classic German concept of "competition of antigens" and demonstrated clearly that mixtures of bacterial vaccines and toxoids might be used. Furthermore, he showed that the immune response to the toxoids, that is, antitoxin production, was potentiated thereby. Such multiple antigens, usually incorporating typhoid vaccine, have been employed by the French Public Health officials for more than ten years. In 1936 Bordet, the discoverer of the pertussis bacillus, reported favorably on a mixture of pertussis vaccine and diphtheria toxoid.[29] More recently reports on mixed immunization have appeared in this country.[30]

In 1941 a trial of combined immunization was begun in the Children's Clinic at Stanford. A mixture of aluminum hydroxide adsorbed diphtheria and tetanus toxoids containing twenty billion H. pertussis per cubic centimeter was used.[31] Only two injections of 1 cc. each were given. An interval of three months was used. Table 1 details the results of the serologic observations. The determinations here recorded apply only to children who should have but did not have any reinjections of any kind. Hence the antitoxin and antibody levels indicate the serologic immunity following the basic course of only two injections plus the slight added stimulus of a Schick test six months after the basic course.

TABLE 1.—*Schick Tests, Tetanus Antitoxin Determinations and Pertussis Agglutination Reactions in Children Who Received Two Injections of Combined Antigens*

	Interval After Second Injection		
	3 to 6 Mo.	7 to 13 Mo.	2½ to 3¾ Yrs.
Negative Schick tests	114	..	24
Positive Schick tests	0	..	0
Tetanus antitoxin			
0.1 U./Cc. or more	86	72	34
Less than 0.1 U./Cc.	1	1	0
H. pertussis agglutinins			
Present in dilution of			
1:320 or higher	52	41	8
1:160 or lower	18	21	10
Absent	0	4	4

Each injection of combined diphtheria-pertussis-tetanus antigens, aluminum hydroxide adsorbed ("D-P-T Alhydrox," Cutter Laboratories), equals 1 cc.; the interval equals three months.

TABLE 2.—*A Conservative Schedule for Office Immunization Procedures Planned to Obtain Optimal Time Relationships for Basic Courses and Reinjections*

Age	Injection
6 mo.	H. pertussis vaccine
7 mo.	H. pertussis vaccine
8 mo.	Mixed diphtheria and tetanus toxoids containing H. pertussis vaccine
10 mo.	Smallpox vaccine
1 yr.	Mixed diphtheria and tetanus toxoids
15 mo.	Tetanus toxoid; Schick test
18 mo.	H. pertussis vaccine
2 yrs.	Tetanus toxoid
4 yrs.	Tetanus toxoid
6 yrs.	Smallpox vaccine; H. pertussis vaccine; Schick test; tetanus toxoid
Thereafter continue tetanus toxoid every 2 yrs.	

It is apparent from the table that a satisfactory immune response was obtained with respect to diphtheria. Although the numbers are small the persisting uniform Schick negativity is gratifying. The tetanus antitoxin produced is rather remarkable. To my knowledge uniform levels of 0.1 unit or more after two and a half years have not been previously described. (This study will be reported in more detail elsewhere.) On the other hand, the pertussis agglutination titers are not satisfactory. In my experience children carrying

24. (*a*) Sako, W.; Treuting, W. L.; Witt, D. B., and Nichamin, S. J.: Early Immunization Against Pertussis with Alum Precipitated Vaccine, J. A. M. A. **127**: 379 (Feb. 17) 1945. (*b*) Sako, W.: Studies on Pertussis Immunization, J. Pediat. **30**: 29, 1947.

25. Sauer, L. W.: The Age Factor in Whooping Cough, Am. J. Path. **15**: 719, 1941. Faber, H. K.: Personal communication to the author. Sako.[24b]

26. Singer-Brooks, C.: Pertussis Prophylaxis, J. A. M. A. **114**: 1734 (May 4) 1940. Cravitz, L., and Cauley, J. H.: Pertussis Immunization Program of the Boston Health Department, ibid. **129**: 539 (Oct. 20) 1945.

27. Shaw, E. B.: The Prevention of Pertussis by the Use of Bacterial Vaccines, J. Pediat. **17**: 414, 1940. Lapin, J. H.: The Stimulation Dose in Whooping Cough, ibid. **20**: 18, 1942.

28. Ramon, G.: Mixed Vaccinations, Internat. Clin. **1**: 241, 1939.

29. Bordet, J., cited by Ramon.[28]

30. Simon, H., and Craster, C. V.: Simultaneous Immunization with Combined Diphtheria-Whooping Cough Vaccine, J. M. Soc. New Jersey **38**: 461, 1941. Kendrick.[33b] Daughtry-Denmark.[33a] Sauer, L. W.; Tucker, W. H., and Markley, E.: Immunity Responses to Mixtures of Diphtheria Toxoid and Pertussis Vaccine, J. A. M. A. **125**: 949 (Aug. 5) 1944. Lapin, J. H.: Mixed Immunization in Infancy and Childhood, J. Pediat. **22**: 439, 1943. Miller and others.[18] Hamilton, P. M., and Knouf, E. G.: Combined Immunization Against Diphtheria, Tetanus and Pertussis, J. Pediat. **25**: 236, 1944.

31. Supplied by Cutter Laboratories.

agglutinin in high titer (1:320 or more) are protected.[32] With lower titers they may or may not be. The levels here found are inferior to those observed in the past following 80 billion H. pertussis in isotonic solution of sodium chloride. Furthermore, 2 children in this group were reported to have had whooping cough.

In the light of the above observations the clinic immunization schedule has been modified by the insertion of a dose of pertussis vaccine midway between the two triple antigen injections.

Some such schedule of combined immunization is of particular value in public health quarters and in crowded dispensaries. In private practice the use of mixtures may often be found a convenience. Their principal disadvantage is not the slightly more pronounced local reactions but the difficulty involved in obtaining the optimal interval for each ingredient in the mixture. In private practice optimal time relationships need not be compromised when the child is being examined monthly. A conservative schedule for office immunization procedures is submitted in table 2. This schedule is based on my conclusions as to optimal time relationships for basic courses and reinjections.

CONCLUSIONS

For primary (basic) active immunization of infants the following preparations are recommended: against smallpox, calf lymph virus; against diphtheria and tetanus, alum precipitated toxoids or aluminum hydroxide adsorbed toxoids, and against pertussis, phase 1 H. pertussis vaccine in isotonic solution of sodium chloride or alum precipitated.

Frequent reinjections of tetanus toxoid are obligatory to maintain immunity. Reinjections of pertussis vaccine and diphtheria toxoid are important, as is also smallpox revaccination.

Combined immunization with mixtures of toxoids and H. pertussis vaccine is valuable in public health and dispensary practice. Combined immunization may often be a convenience in private practice.

ABSTRACTS OF DISCUSSION

Dr. Hulda E. Thelander, San Francisco: Although considerable attention is given to the immunizing agent little is given to the host or recipient of immunization, who probably is more variable than is the agent used. It is generally recognized that characteristics of the human race such as height, weight and intelligence quotient vary widely; when persons are arranged according to their measurements they will fall into typical variability curves. Immunity, or the capacity to be immunized, however, is an intangible characteristic which does not lend itself to easy measurement. Years ago, in the study of antitoxin levels after immunization it was considered that it would be desirable to demonstrate graphically that immunity, like other factors, showed variability. Difficulty arose, however, when the figures spread from 0.005 unit to 50 units per cubic centimeter of serum. Finally, by arranging the figures by multiples of ten, it was possible to plot a variability curve. The children were immunized by one of the standard methods, but the adults were given 0.1, 0.2 and 0.2 cc. at two week intervals, and severe reactors were continued at 0.1 cc. instead of increasing the immunization dosage to 0.2 cc. The curves for children and adults were similar. The object lessons derived from the study were two: first, that the relatively few persons who resist immunization are the ones to whom special attention must be given, such as larger doses, more injections and frequent boosters; second, that it is unnecessary to subject the majority of adults to the undesirable effects of large doses of toxoid, since most persons immunize as well with much smaller doses.

Dr. Alfred E. Fischer, New York: Every pediatrician must look forward to the time when his young patients will no longer scream with terror when they approach his office. The earlier age of injection and a decrease in the number of injections will go far to make the practice of pediatrics more tolerable. The morbidity rate for pertussis indicates the need for early immunization. Until recently it had been assumed, largely from animal experimentation, that the young infant was unable to produce antibodies effectively. Then Sako showed that if adequate amounts of phase I pertussis organisms were injected a good agglutination titer could be obtained in infants 4 to 8 weeks old. Moreover, his clinical results were satisfactory; fewer of the injected infants contracted pertussis on subsequent exposure than did controls. Furthermore, the immune response persisted. Recently Peterson, in as yet unpublished experiments, showed that infants less than 3 months of age produced adequate antibodies against pertussis and tetanus. On the other hand, the antitoxin level produced against diphtheria was not as high as that produced in older infants. Several groups of workers in New York are studying different combinations of antigens in various age groups. From the data at hand, one cannot as yet recommend a standardized procedure using triple antigens, that is pertussis, diphtheria and tetanus. Needs might vary in different communities and even in the same community from year to year. Since there has been only 1 infant less than 6 months of age with diphtheria, and only 9 under 1 year of age at Willard Parker Hospital in the past seven years, there is no necessity for extremely early immunization against this disease in New York at present. Nor is tetanus immunization important in the young infant in this city; however, since it can be combined so readily with the diphtheria injection, one may as well administer the two toxoids together. Since active immunity tends to weaken after a time, the administration of a stimulating dose of antigen should become a well established procedure. The triple antigen in doses of 0.2 or 0.3 cc. would seem to be excellent for this purpose in a previously immunized infant, one year after basic immunization, as Dr. Miller has suggested, and perhaps every two years thereafter. A suggested immunization schedule is as follows: 4 months of age, smallpox vaccination (spring or fall); 5 months, first pertussis; 6 months, diphtheria—tetanus—pertussis; 7 months, diphtheria—tetanus—pertussis; 8 months, smallpox vaccination, if not done at 4 months; 9 months, diphtheria and tetanus; 14 months, Schick test; 2 years, diphtheria—tetanus—pertussis (stimulating injection); and two or three years later, a stimulating injection. Young infants tolerate smallpox vaccination well, so I can see no objection to vaccinating a healthy infant at 3 or 4 months of age except in extremely cold or hot weather. Early pertussis immunization is desirable, so the first of such injections can be started at 5 months of age.

Dr. John A. Toomey, Cleveland: I am chairman of that committee. Members of the committee are scattered over the United States, and there exist many different opinions about any procedure, however simple. Frequently, the committee has to stay put for a period of time before it can get even a semblance of agreement on a particular point. Consequently, the official opinion of the committee must of necessity lag a little behind the more decided advances suggested by Drs. Miller and Thelander. An opinion given by the committee must be on a point which is tried and universally accepted. That doesn't mean to say that the opinion is always right. For the past two years I have been on a national committee that has been studying the effect of multiple antigens. It first used five antigens. I won't go into this report because it is to be published later by the committee as a whole, but I will give the general substance of it. Five antigens were tried, including typhoid, scarlet fever and the three antigens reported on this morning. The reactions were severe, and even the persons on whom the

32. Miller, J. J.; Silverberg, R. J.; Saito, T. M., and Humber, J. B.: An Agglutinative Reaction for H. Pertussis: Its Relation to Clinical Immunity, J. Pediat. 22: 644, 1943. Sako and others.[24]

antigens were tried, patients in an epileptic and mental institution, even those patients complained. Subsequently, it was found that the scarlet fever antigen was causing the trouble. The three antigens suggested by Drs. Miller and Thelander, tetanus, diphtheria and pertussis, have been tried by a national committee; after two years' work, their results will be reported by this committee at the American Public Health Conference in November, 1946. I agree with Dr. Thelander that there is no reason why adults should be immunized, especially against diphtheria, and also I agree with her that adults can be immunized with extremely small doses. There is some objection to the routine use of tetanus antigens. Two members of the committee of which I am chairman do not believe that it should even be used, but the majority feel that there is no objection to the use of the three antigens. I wondered if Dr. Miller could give some information as to whether he uses combinations in the subsequent booster dose. For instance, if he used the triple antigen and then subsequently has a child exposed to tetanus, will he give a booster injection of an antigen composed of two materials (diphtheria and tetanus) or will he use a single antigen, that is, a tetanus antigen? It has been said by some that when the mixed antigens have been used in the booster dose there have been a few reactions of an anaphylactic nature. I would like to ask him about that.

DR. ERNEST WOLF, San Francisco: Whereas it is desired to prevent disease in childhood, one should at the same time be extremely careful that the relation between patient and physician also be saved. I make a great plea to all who make those schedules to minimize the number of injections necessary between the sixth and eighteenth month. At this age children are easily upset and are very much aware of what is done to them. Should it be proved that measures taken in the first four or five months give sufficient immunity to prevent later troubles, this would be the period of choice from the point of view of mental hygiene and patient-physician relations. When I see Dr. Miller's schedule of seven injections for infants between 7 and 15 months of age, I am a little afraid that pediatricians will have still more howling in their offices.

DR. JOHN MILLER JR., San Francisco: In answer to Dr. Toomey's question What agents are used for booster injections? circumstances dictate the choice. If a child is brought in with a wound, he is injected with 1.0 cc. of fluid tetanus toxoid regardless of the type of tetanus toxoid he received in the past. If a child has been exposed to whooping cough a booster injection of 20 billion H. pertussis organisms in isotonic solution of sodium chloride is given. As speed in raising antitoxin or antibody titer is desired, adsorbed preparations are not the agents of choice. On the other hand, for routine reinjections at 6 years of age a mixture of antigens is employed. H. pertussis vaccine mixed in the syringe with adsorbed tetanus toxoid is given. If the Schick test done at this visit becomes positive I then give the child diphtheria toxoid alone. As I mentioned previously, I am guided in my dosage by the child's reaction to the Schick control test of diluted toxoid. I do not give mixtures containing diphtheria toxoid to the child 6 years of age unless I know that the child is not sensitive to diphtheria toxoid.

Cardiac Lesions in Rheumatoid Arthritis.—We used to be taught, even until the last year or two, that rheumatoid arthritis was wholly different from and not in any way related to rheumatic fever. These postmortem studies show that this is not so; they reveal that, in a high percentage of cases of chronic arthritis, changes are found in the heart which are wholly indistinguishable from the changes in the heart in cases of rheumatic fever. It really is astounding when we think that postmortem examinations by reliable pathologists show that 30 to 60 per cent of patients dying with rheumatoid arthritis have cardiac lesions, valvular lesions of the heart that are identical in every way with the valvular lesions found in rheumatic fever. —Bohan, Peter T.: The Concomitant Diseases or Conditions Associated with Polyarthritis, *Postgraduate Medicine*, May 1947.

PULMONARY ARTERIOVENOUS ANEURYSM WITH SECONDARY POLYCYTHEMIA

Report of the First Case Treated by Lobectomy

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In 1938 the attention of the members of the medical profession was first called by Rodes [1] to congenital pulmonary arteriovenous aneurysm with secondary polycythemia as a cause of cyanosis. Since this first report only 7 of these cases have been reported,[2] although 2 others without polycythemia have been noted,[3] and rupture of the arteriovenous fistulas has been recorded as a cause of death in infants.[4] In none of the 7 cases just mentioned [2] was the condition diagnosed correctly by the first physician to see the adult patient. As 3 of these patients were completely cured by total pneumonectomy [5] and as the lesion has been recognized more frequently in the last eight years, it is important that physicians become aware of this condition as a cause of cyanosis.

Since our patient is the first to be treated by lobectomy and since this curable condition is so seldom diagnosed, we wish to report our case and review the diagnostic features of this lesion.

REPORT OF CASE

History and Preoperative Data.—M. M., a white unmarried woman aged 27, was first seen in the medical outpatient clinic of the University Hospital, Ann Arbor, Mich., on Oct. 26, 1945 with the chief complaint of "blueness." Mild cyanosis had been noted since birth but had increased in intensity in the last two years. She stated that it had occasionall, decreased to almost complete disappearance for intervals as long as two weeks. From birth till the age of 9 years she had had moderate epistaxes once a week. After the age of 9 years these episodes decreased in frequency. She had also been bothered since her earliest memory by the prompt appearance of paresthesias in the extremities on the left side when she would lie on this side. Three or four years before admission she had noted the onset of episodes of syncope (lasting about three minutes), dizziness and paroxysmal nocturnal dyspnèa occurring about once a month.

In September 1945 she consulted her physician, Dr. Cecil Corley of Jackson, Mich., because of pain in the legs extending from the feet to the hips and present since birth. Dr. Corley noted the cyanosis and obtained roentgenograms of the chest which showed no evidence of congenital heart disease but did show a tumor in the right lower lung field. He then referred her to the University Hospital, Ann Arbor, Mich., for diagnosis and treatment.

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1. Rodes, C. B.: Cavernous Hemangiomas of the Lung with Secondary Polycythemia, J. A. M. A. **110**: 1914 (June 4) 1938.

2. (*a*) Adams, W. E.; Thornton, T. F., Jr., and Eichelberger, L.: Cavernous Hemangioma of the Lung (Arteriovenous Fistula), Arch. Surg. **49**: 51 (July) 1944. (*b*) Hepburn, J. H., and Dauphinee, J. A.: Successful Removal of Hemangioma of the Lung Followed by the Disappearance of Polycythemia, Am. J. M. Sc. **204**: 681, 1942. (*c*) Goldman, A.: Cavernous Hemangioma of the Lung: Secondary Polycythemia, Dis. of Chest **9**: 479, 1943. (*d*) Jones, J. C., and Thompson, W. P.: Arteriovenous Fistula of Lung: Report of Patient Cured by Pneumonectomy, J. Thoracic Surg. **13**: 357, 1944. (*e*) Smith, H. L., and Horton, R. T.: Arteriovenous Fistula of the Lung Associated with Polycythemia Vera: Report of a Case in Which the Diagnosis Was Made Clinically, Am. Heart J. **18**: 589, 1939. (*f*) Makler, P. T., and Zion, D.: Multiple Pulmonary Hemangiomata, Am. J. M. Sc. **211**: 261, 1946. (*g*) Rodes.[1]

3. (*a*) Sisson, J. H.; Murphy, G. E., and Newman, E. V.: Multiple Congenital Arteriovenous Aneurysms in the Pulmonary Circulation, Bull. Johns Hopkins Hosp. **46**: 93, 1945. (*b*) Janes, R. M.: Multiple Cavernous Hemangiomas of the Lungs Successfully Treated by Local Resection of the Tumors, Brit. J. Surg. **31**: 270, 1944.

4. Bowers, W. F.: Rupture of Visceral Hemangioma as Cause of Death, Nebraska M. J. **21**: 55, 1936.

5. Jones and Thompson,[2d] Smith and Horton.[2e] Sisson, Murphy and Newman.[3a]

The intake of drugs by the patient in the past had been limited to one or two tablets of acetanilid as often as once a month. Of significance in the family history was the fact that the patient's mother and living brother had had frequent epistaxes all their lives. The mother was described as having been exactly comparable to the patient in her degree of physical

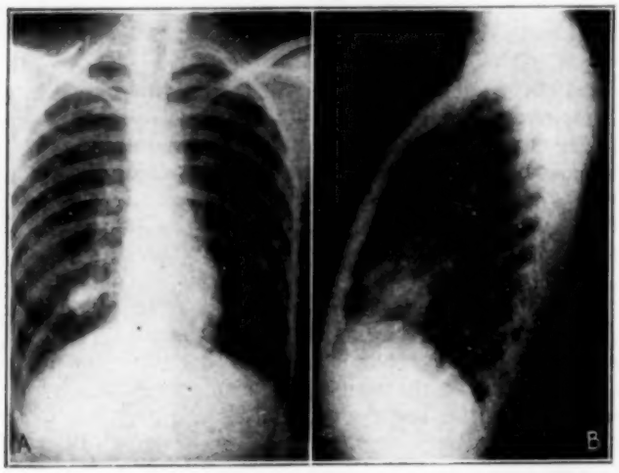

Fig. 1.—*A*, preoperative roentgenogram showing the arteriovenous aneurysm in the right lower lung field; *B*, right lateral view.

underdevelopment and cyanosis. She died at the age of 37 of "pneumonia at childbirth." When the brother was 12 years of age he was told by his physician that he had an incurable "rheumatic heart." He is apparently well now at the age of 30 and is not cyanotic.

On physical examination the patient was found to be 5 feet 2 inches (158 cm.) tall and to weigh 89 pounds (40.4 Kg.). She was poorly nourished and poorly developed, with masculine distribution of body hair and hypoplastic breasts. Her blood pressure was 110 systolic and 80 diastolic in the right arm, in the reclining position, while the blood pressure in the left arm could not be obtained. The radial pulses were strong and equal bilaterally. The patient exhibited generalized cyanosis, most pronounced over the face, lips and hands. In addition close inspection revealed numerous pinhead-sized to pinpoint-sized blue spots, which appeared to be small, superficial hemangiomas, on the face, lips and palms. The lung fields and heart were entirely normal to physical examination. The spleen was not enlarged. The extremities showed only minimal clubbing of the fingers. The superficial veins were unusually prominent. The rest of the physical examination was entirely noncontributory.

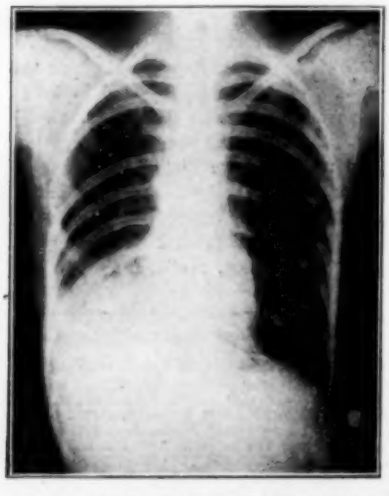

Fig. 2.—Postoperative roentgenogram of the chest, with aneurysmal communication removed with lower lobe of the right lung.

Laboratory examinations included a complete blood count with a hemoglobin level of 21.6 Gm. (138 per cent). The red blood cell count was 8.2 million per cubic millimeter with a hematocrit reading of 75 volumes per cent. The mean corpuscular volume was 91 cubic microns. The white blood count was 7,000 per cubic millimeter with a normal differential. The sedimentation rate was 1 mm. per hour by the Wintrobe method. Urinalysis and the Kahn test on the blood were negative. These findings together with the roentgenograms sent by Dr. Corley suggested the diagnosis of pulmonary arteriovenous aneurysm with secondary polycythemia.

The patient was advised to have a lobectomy as treatment for her disease. She returned on March 18, 1946, at which time the history, physical examination and laboratory studies were not significantly changed. Roentgenologic studies showed a 3 cm. area of increased density in the medial portion of the right lower lung field, half way between the anterior and posterior surfaces of the lung. Pulsations were definite but very small. A tail-like projection seemed to extend from this area of increased density to the hilar vessels on that side (fig. 1). An electrocardiogram, read by Dr. F. N. Wilson, was well within normal limits. Oxygen saturation studies done by Dr. Vivian Iob showed the arterial blood saturation, based on hemoglobin content, to be 77 per cent and the venous oxygen saturation to be 61 per cent. The arm to tongue

Fig. 3.—The aneurysm, sectioned, with arterial side on the right and venous side to the left. The photograph was supplied by Dr. C. V. Weller, chairman of the Department of Pathology.

(dehydrocholic acid) circulation time was 12 seconds, the normal being 10 to 16 seconds. The vital capacity was 2,000 cc.

Operative Observations.—On March 21, 1946 a lobectomy of the lower lobe of the right lung was performed by one of us (F. X. B.) under intratracheal ether vapor and oxygen anesthesia. With the patient in the left lateral decubitus position the incision was made along the course of the fifth rib. The rib was resected subperiosteally. Although lobectomy was contemplated (we were under the impression that the aneurysm lay in the posterior aspect of the middle lobe) the fifth rib was resected rather than the seventh because we were aware of the experience of Jones and Thompson [2d] who had planned a lobectomy of the upper lobe for their patient but were forced to perform a pneumonectomy because of a vascular abnormality (absence of the inferior pulmonary vein). On opening the pleura no abnormalities were observed, and on separating the fissure between the middle and lower lobes the aneurysm was immediately apparent on the anterior surface of the lower lobe of the lung, with its upper margin approxi-

mately 6 cm. from the hilum of the lobe. The aneurysm itself measured about 4 cm. in diameter, was dark red and so thin-walled that the blood could be seen passing through it. There was no visible pulsation, and no thrill was palpable.

The entire fissure surface of the lower lobe, superior to the aneurysm, was covered with a fine meshwork of telangiectatic vessels, which made dissection of the hilum somewhat more hemorrhagic than is usual. After it had been ascertained that both an inferior and superior pulmonary vein were present, the lower lobe of the right lung was resected by the individual dissection and ligation technic. The fissure between the upper lobe and the apex of the lower lobe was incomplete, division being accomplished between hemostats with oversewing of the cut surface of the upper lobe. Postoperatively the chest was drained by means of an intercostal catheter for forty-eight hours.

The arterial and venous paths to the aneurysm could be demonstrated in the resected lobe (fig. 3).

Postoperative Course.—The postoperative course was uneventful, one thoracentesis being necessary after removal of the drainage tube.

Immediately after the operation there was a noticeable decrease in cyanosis even though polycythemia was still present. During the subsequent hospital days progressive diminution of the cyanosis occurred. The hemoglobin content was 17.5 Gm. on the fourth postoperative day. On the eleventh postoperative day studies of the venous and arterial blood were carried out. Data on the arterial blood were as follows: hematocrit reading 57.7 volumes per cent, hemoglobin 17.9 Gm., oxygen content 21.8 volumes per cent, oxygen saturation 23.7 volumes per cent and the per cent of oxygen saturation had risen to 92.2 per cent (as compared with the preoperative value of 77 per cent). The percentage of venous oxygen saturation was 60. The patient was discharged on April 3, 1946, the thirteenth postoperative day.

On May 1, 1946 she returned for a follow-up examination. She stated that she no longer had leg pain and felt well except for some residual thoracic discomfort. Examination revealed the physical signs incident to lobectomy, mild cyanosis and generalized desquamation of the skin. The blood pressure was now obtainable in the left arm and was 110 systolic and 78 diastolic. Her hemoglobin level on this visit was 18.4 Gm. (118 per cent), as compared with the preoperative level of 21.6 Gm. (138 per cent). Similarly the hematocrit reading was 62.5 volumes per cent and the red blood cells count was 6.7 million, as compared with the preoperative levels of 75 volumes per cent and 8.2 million.

In June roentgenograms of the chest showed a satisfactory operative result and did not disclose other aneurysms (fig. 2).

COMMENT

The clinical picture in this case closely resembles that of the 7 previously reported cases of pulmonary arteriovenous aneurysm with secondary polycythemia. Seven of the 8 patients, including ours, have been aged 20 to 27 years, the other 1 being 40 years old.[2e] Five have been men and three women. The chief complaint on admission has varied so widely as to be of little aid in making the final diagnosis. The symptoms, however, have been remarkably constant and consist of (1) cyanosis, (2) clubbing of the digits and (3) symptoms of anoxia.

1. *Cyanosis.*—Half of the patients had exhibited some degree of cyanosis since birth. The others showed cyanosis for two to sixteen years.[6] It is notable that cyanosis secondary to a pulmonary arteriovenous aneurysm can occur in the absence of polycythemia.[2]

2. *Clubbing of the Digits.*—Clubbing of at least the fingers was present in all cases. The degree of clubbing roughly paralleled the size and number of the pulmonary fistulas. Our patient had but minimal clubbing secondary to one 3 by 2 cm. fistula.

3. *Symptoms of Anoxia.*—The most commonly observed symptom of anoxia was dyspnea, which was present in 6 of the 8 cases.[7] Only cyanosis was noted by Jones and Thompson.[2d] Other common symptoms of anoxia manifested by the majority of patients were mild or decided syncope with or without convulsions, paroxysmal nocturnal dyspnea, substernal oppression and paresthesias. These symptoms of anoxia are probably secondary to the extremely low oxygen saturation of arterial blood observed in patients with this condition.

A peculiar and unexplained aspect of symptomatology in 3 of the cases is unilateral complaints related to the left side of the body. One patient[2a] could not lie on the left side because it caused him to cough. This symptom was relieved by removal of the fistula in the left lung by left pneumonectomy. Our patient could never lie on her left side because the extremities on the left would immediately develop intense paresthesias. This complaint disappeared when the fistula on the right was removed by lobectomy of the lower lobe of the right lung. In another patient[3a] there developed numbness in the left arm and shoulder and weakness of the left hand shortly before admission to the hospital. Unfortunately we do not know the effect of treatment in this case, since the patient died during angiography.

The family history of heredity of this lesion is interesting. In 4 instances this point was omitted[8] and in 2 the presence of cyanosis in other members of the family was denied.[9] In the case of Adams and his associates[2a] the father of the patient had had blue spots on his lips and nose bleeds similar to those of the patient. This was also true of other men in the family. The father had had a roentgenogram of the chest which had shown "shadows." The description of our patient's mother suggests a hereditary tendency. The brother's history is suggestive and is being investigated at the present time.

The only constant observations on physical examination in the reported cases were cyanosis and clubbing of the fingers. Strangely enough, only 3 patients had a murmur produced by the vascular communication.[10] In the majority of the reports no pulsation of the communication was noted by roentgenologic examination. No cardiac abnormality or abnormality of the pulse pressure or of the pulse rate was present. Five of the 8 patients were underdeveloped, presumably due to the prolonged anoxia caused by the congenital lesion.[11] Five of the 8 patients had cutaneous hemangiomas.[12] These were located on the face, on the lips, behind the ear and on the palms. Since they easily pass unnoticed, it is entirely possible that all patients with this type of lesion have had them. The surface of the lung at surgical intervention or autopsy is also frequently covered with these small hemangiomas. Since the

6. Adams, Thornton and Eichelberger.[2a] Hepburn and Dauphinee.[2b] Goldman.[2c] Makler and Zion.[2f]

7. Rodes.[1] Hepburn and Dauphinee.[2b] Goldman.[2c] Smith and Horton.[2e] Makler and Zion.[2f]

8. Hepburn and Dauphinee.[2b] Jones and Thompson.[2d] Smith and Horton.[2e] Makler and Zion.[2f]

9. Rodes.[1] Goldman.[2c]

10. Jones and Thompson.[2d] Smith and Horton.[2e] Makler and Zion.[2f]

11. Rodes.[1] Hepburn and Dauphinee.[2b] Goldman.[2c] Smith and Horton.[2e]

12. Rodes.[1] Adams, Thornton and Eichelberger.[2a] Goldman.[2c] Makler and Zion.[2f]

spleen is never enlarged, with the possible exception of the case of Goldman [2e] in which it was "just palpable," and since the white blood count is never elevated, the possibility of polycythemia vera may be eliminated. In spite of this fact the disease of 2 patients [13] with pulmonary aneurysms was so diagnosed and treated.

Most striking of laboratory observations is the secondary polycythemia with red blood cell counts ranging from 6.5 million [2e] to 11.45 million.[2c] The hemoglobin level in these cases ranged from 118 per cent to 138 per cent. The highest hematocrit reading recorded [2b] was 80 volumes per cent. The oxygen saturation of the arterial blood ranges from 70 to 80 per cent. The vital capacity and the blood circulation times are always within the normal range, although the latter approaches the lower limit of normal.

In none of the 8 cases was the diagnosis made by the first physician to see the patient. In 3 cases more fistulas were found at pathologic examination than by roentgenologic examination.[14] Yet surgical extirpation of the main fistulous communication [2a] has abolished the symptom complex. Apparently the most frequent mode of death is by pulmonary hemorrhage [15] as in pulmonary arterial aneurysms. The fact that patients treated have been reported to be cured at five and a half months,[2a] one year [2b] and seven months [2d] after pneumonectomy suggests that surgical treatment is worth while. Pneumothorax on the side of the lesion is worthless either as a diagnostic or therapeutic procedure, since no change in the clinical picture occurs even though the lung is thoroughly collapsed.[2b]

SUMMARY

Cyanosis secondary to congenital pulmonary arteriovenous aneurysm with secondary polycythemia is one type of congenital cyanosis that can be completely cured by surgical intervention. Although this lesion is supposedly extremely rare, the fact that all 8 cases have been diagnosed since 1938 suggests that many of these lesions have been missed in the past.

The most important prerequisite for making the diagnosis is an awareness of this lesion as a cause of cyanosis and polycythemia. The clinical picture consists of a patient of either sex, usually from 20 to 30 years of age, with cyanosis recently increased in intensity. Symptoms of anoxia predominate, with hemorrhage from superficial hemangiomas, usually from the nose. Cyanosis has often been present in siblings or parents. Physical examination reveals clubbing of the digits and cyanosis. Maldevelopment and superficial hemangiomas are also present, but there is usually no abnormality of the heart, pulse pressure or pulse rate and no pulmonary vascular murmur.

The vital capacity and blood circulation times are normal, but secondary polycythemia is present. The diagnosis is clinched by the roentgenologic observation of tumor-like shadows in the lung. These shadows usually do not pulsate but do usually show communications with the hilar vessels. The patient is completely cured by pneumonectomy, lobectomy or local excision of the fistula.

13. Jones and Thompson.[2d] Smith and Horton.[2c]
14. Rodes.[1] Adams, Thornton and Eichelberger.[2a] Sisson, Murphy and Newman.[2a]
15. Rodes.[1] Bowers.[4]

THE MECHANISM AND MANAGEMENT OF INJURIES OF THE HEAD

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Great strides have been made in the study of the mechanism and management of injuries of the head during the past ten years. In the present paper it is proposed that the more important of these studies be analyzed.

MECHANISM OF CRANIOCEREBRAL TRAUMA

Injury to the head may occur by direct or indirect violence. In a direct blow the head is relatively fixed and the striking object is in motion. A direct blow may cause compression of the intracranial contents by deformation of the skull, and it may set the head to motion (acceleration). In indirect blows the head in motion comes in contact with a nonmoving or slower moving object. Under these circumstances, compression and deceleration may obtain. Compression, deceleration and acceleration frequently coexist in the usual injuries such as auto accidents, falls, etc.[1] With direct blows the lesion is more likely to be limited to the head, whereas with indirect violence the injuries are usually multiple.

Deformation of the skull and acceleration of the head result in increased intracranial pressure waves, with steep gradients. The presence of the craniospinal junction at the foramen magnum may be an all-important factor in the involvement of centers in the brain stem. Such an opening may cause pressure gradients between the cranial cavity and the spinal canal, with encroachment on medullary structures.

The effects of craniocerebral trauma in the experimental animal are manifested by alterations of function of centers in the brain stem, namely the vasomotor, the respiratory and the sensory-motor centers supplying the face. The generalized body rigidity frequently seen results from stimulation of the pyramidal tracts in the brain stem.[2] Besides these disturbances in the brain stem, hemispheric abnormalities have been demonstrated by the electroencephalograph, which has shown increase in amplitude and frequency of pattern immediately following impact in some experiments.[3] In others, there has been an initial depression of the electroencephalographic pattern.[4] It is proved beyond doubt that death from acute injury of the head in the experimental animal results from direct damage to medullary centers. Autopsies in human beings found dead at the scene of accident reveal pathologic changes in the form of petechial hemorrhages in the brain stem similar to those observed in the experimental laboratory.

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2. Polis, A.: Rev. chir., Paris **14**: 274 and 645, 1894. Denny-Brown, D., and Russell, W. R.: Brain **64**: 93, 1941. Gurdjian, E. S., and Webster, J. E.: Surgery **16**: 381, 1944.
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It is reasonable to assume that in the human being death soon after injury is due to medullary paralysis, as it has been found to be in the experimental animal.

The effects of trauma of the head can be correlated with the amount of absorbed energy whether the head is fixed or free to move. In the experimental animal (dog), 200 inch-pounds of energy absorbed by the head in $\frac{1}{1,000}$ to $\frac{2}{1,000}$ of a second or less, causes profound pathophysiologic effects, usually terminating fatally. When the head is free to move, the possibility of mass movements of the brain is greater.

The definition of the term "concussion," or immediate post-traumatic unconsciousness "unassociated with microscopic or macroscopic lesions of the brain," should be changed in the light of recent experimental work.[5] New pathologic technics have revealed the presence of intracellular changes even in the most minimal degrees of concussion. The damage to the cells may or may not be reversible.

We have had the opportunity to study deformations of the skull and changes in intracranial pressure at the time of impact by certain new electronic and engineering technics. For the study of deformations of the skull, we have used (1) the strain gage–cathode ray oscilloscope method and (2) the "stresscoat" technic. The former is a sensitive, quantitative method of determination of deformation; the latter permits the study of the direction and the path of strains following a blow.

Strain Gage–Cathode Ray Oscilloscope Method.—The strain gage is a length of wire $\frac{1}{1000}$ inch (0.0254 mm.) in diameter which is cemented to the surface of the skull with methyl methacrylate cement.[6] Deformations of the skull following a blow also deform the strain gage. Inbending of the skull causes a shortening of the wire, whereas outbending results in a lengthening of the wire in the strain gage. Shortening of the wire results in decrease in the resistance, and lengthening, an increase in the resistance through the gage. Such changes in resistance, due to deformations of the skull, are shown on the oscillograph, and permanent records are obtained by photography.

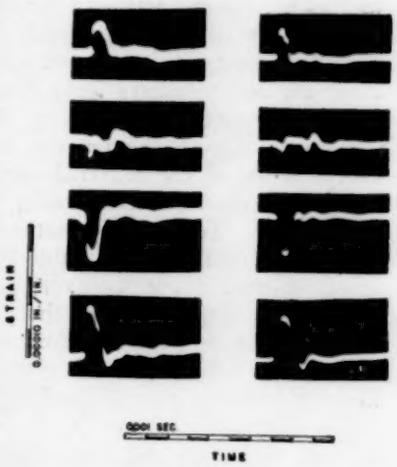

Fig. 2.—To the left are records of one human autopsy specimen and to the right are records of another specimen. The blows were administered as indicated in figure 1. Upper row, hammer blow at point 1 with recording at strain gage A; second row, blow at point 2 and recording at strain gage A; third row, blow at point 1 and recording at strain gage B; fourth row, blow at point 2 with recording at strain gage B. Upswing denotes tension, and downswing denotes compression. Total time of disturbance was $\frac{1}{250}$ of a second. Initial deformation time was $\frac{1}{2,000}$ to $\frac{1}{5,000}$ of a second. There were three to six oscillations during the disturbance at the rate of 900 to 1,200 cycles per second.

The measurements with the strain gage of deformations in the skull of the dog under pentobarbital sodium anesthesia were similar to those obtained twenty-four hours after the death of the animal with intracranial contents intact. The dry skull is not suitable for this study, owing in part to the absence of the dampening effect of the intracranial contents. Since strain gage measurements of deformations of the skull were known to be the same in the living under pentobarbital sodium anesthesia and in the dead dog with intracranial contents intact, human autopsy material, soon after death, was studied with this technic. It was concluded that the findings in the autopsy specimens represent conditions present in the living (fig. 1).

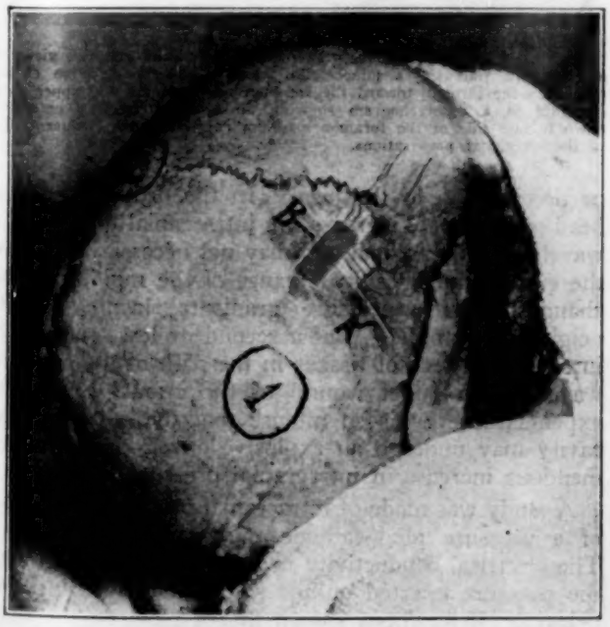

Fig. 1.—A human skull prepared for strain gage studies of deformation from hammer blows; 1 and 2 are the areas of impact, and A and B are the strain gages cemented to the surface of the skull. The records obtained from such preparations are seen in figure 2. Previous experimental work has shown that the strain gage measurements of the dead skull with contents intact are similar to those obtained on the skull of the animal under anesthesia. It is felt, therefore, that these studies on the human skull may represent premortem conditions.

On the delivery of a blow to the human skull, the time of the initial deformation varied from $\frac{1}{5,000}$ of a second to $\frac{1}{2,000}$ of a second. The total elapsed time for the deformation pattern was between $\frac{1}{200}$ to $\frac{1}{250}$ of a second. In different parts of the skull there were 3 to 6 cycles during the deformation process. These occurred at the frequency of about 900 to 1,200 cycles per second (fig. 2).

The pattern of deformation shown by this method is the same irrespective of the energy of the blow. Whether the impact is the result of injury by hammer, brick or fall, the deformation pattern is also the same provided that there is no perforation of the skull from high velocity blows.

"Stresscoat" Studies of Deformations of the Skull.—By the "stresscoat" technic, which employs a strain-sensitive lacquer, the direction and the path of strains were studied. With a lacquer of a given sensitivity applied to the surface of the skull, deformations of the order of 0.0005 to 0.0015 inch per inch resulted in cracks in the lacquer coating. These cracks demonstrate the paths of the strains following the blow.[7] Experimental observations have shown that the deformation patterns in the living animal under anesthesia, in the dead animal with intracranial contents intact and in the dry skull of the same animal are similar.

5. Windle, W. F.; Groat, R. A., and Fox, C. A.: Surg., Gynec. & Obst. **79**: 561, 1944.
6. Gurdjian, E. S., and Lissner, H. R.: J. Neurosurg. **1**: 393, 1944.
7. Gurdjian, E. S., and Lissner, H. R.: Surg., Gynec. & Obst. **81**: 679, 1945.

The magnitude of strain may be different, but the paths and directions are the same.

By a study of freshly obtained skulls, it was found that blows in various regions of the skull caused discrete areas of deformation in the region of impact as well as in remote parts. In the occipital region, cracks appeared in the lacquer with half the amount of absorbed energy as compared with the frontal region. This technic measures only tensile (tearing apart) strains; compression (pushing together) strains are not seen. Up to the present time we have studied the behavior of the external surface of the skull and have no information concerning what may happen on the interior. Tensile deformations on the internal surface of the skull are undoubtedly important and may result in fracture lines. From a study of our present material it appears likely that a certain number of fractures, due to general deformation of the skull, may occur from tension, or an outbending, following the initial inbending, after impact (figs. 3 and 4).

On the basis of our work with the "stresscoat" technic thus far, the following conclusions may be drawn:

1. Threshold deformations in different regions of the skull are obtained with varying energies. For instance, it takes twice as much energy to cause cracks in the frontal as it does in the occipital region.

2. Some fractures may occur from outbending following the initial inbending of the skull.

3. Many fractures occur parallel to lines of stress shown in these studies.

4. From the position and the direction of the skull fracture, one may be able to guess the area of impact which caused the fracture.

5. "Contrecoup" cracks in the lacquer were obtained with occipital blows in 1 specimen. The cracks following an occipital blow in this specimen appeared at the frontozygomaticosphenoid junction on one or both sides

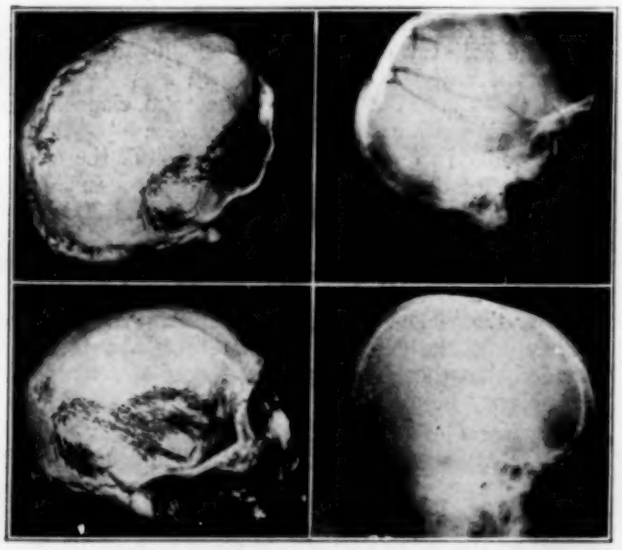

Fig. 3.—To the left, "stresscoat" preparation in a dry skull following a posterior parietal blow. Note that the direction of the cracks in the lacquer are downward and forward toward the temple. To the right are two roentgenograms with fracture lines extending downward and forward toward the temple from the posterior parietal region. The upper roentgenogram also shows a depression of the posterior parietal region at the midline. It is reasonable to assume that the point of impact was in this region, with resultant depression and also fracture lines extending downward and forward toward the temple on both sides, paralleling the stresscoat lines of crack. In the lower roentgenogram the fracture is more widely separated in the parieto-occipital region near the midline. This is the region of the greatest stress from the blow, with the fracture extending downward and forward, eventually becoming a thin line, to disappear in the temple.

Intracranial Changes in Pressure at the Time of Impact.—Increase in intracranial pressure occurs as a result of the deformation of the skull and acceleration

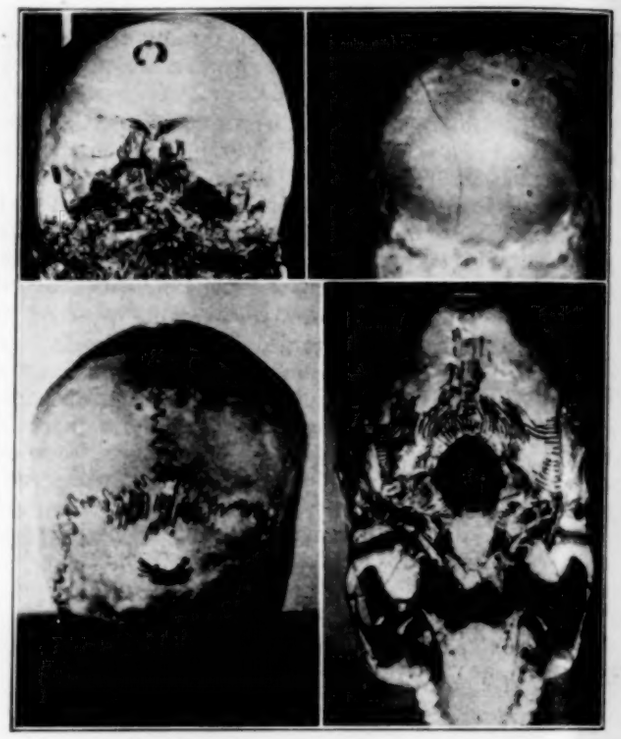

Fig. 4.—"Stresscoat" preparation in a cadaver skull and two views of a dry skull following a midoccipital blow. Note the direction of the cracks in the lacquer toward the foramen magnum. In the upper right quadrant is a roentgenogram showing an occipital fracture extending down to one side of the foramen magnum paralleling the lacquer cracks in the stresscoat preparations.

or deceleration of the head. In the ordinary case of head injury the increase in intracranial pressure is wavelike or directional and may not necessarily involve the entire cranial cavity because of the rapidity of the changes. Steep pressure gradients, lasting in the neighborhood of $\frac{1}{1,500}$ of a second or less, may result in shear and tear of tissues in the path of the pressure wave. In a bullet wound of the cranial cavity the expended energy is so intense that the entire cranial cavity may undergo an explosive effect, due to a tremendous increase in intracranial pressure.

A study was made of intracranial pressure by the use of a pressure plug–cathode ray oscilloscope method. The electrical conductivity in an electrolyte varies with the pressure exerted on the solution.[8] By using this principle, it has been shown that a blow on the skull causes an increase in intracranial pressure at the site of the blow, with the pressure in the negative phase at a point diametrically opposite the area of impact. Working with the experimental animal (dog) it was found that the maximal increase in pressure was obtained in $\frac{1}{1,500}$ of a second following impact.[6] The

8. Fanjung, I.: Ztschr. f. physiol. Chem. **14**: 673, 1894; cited by Creighton, H. J., and Koehler, W. A.: Principles and Applications of Electro-Chemistry, ed. 3, New York, John Wiley & Sons, Inc., 1935, vol. 1, p. 305. Korber, F.: Ztschr. f. physiol. Chem. **67**: 212, 1909; cited by Creighton, H. J., and Koehler, W. A.: Principles and Applications of Electro-Chemistry, ed. 3, New York, John Wiley & Sons, Inc., 1935, vol. 1, p. 305.

period of change in intracranial pressure was in the neighborhood of 1/200 to 1/250 of a second. Three to five oscillations occurred during the period of disturbance.

As a result of the deformation of the skull, the increase in intracranial pressure and the mass movements of the brain, certain primary pathologic conditions ensue. These include cell damage, fracture, contusions and lacerations of brain tissue and intracranial hemorrhage.

CHEMICAL STUDIES IN EXPERIMENTAL HEAD INJURY

The arteriovenous differences in oxygen, carbon dioxide and glucose (samples of blood from the femoral artery and the sagittal sinus) remain within normal limits in moderately and severely injured experimental animals. In the normal brain, chemical studies on specimens frozen with liquid oxygen revealed the presence of 10 to 20 mg. of lactic acid per hundred grams of cerebral tissue.[9] The study of lactic acid in the brain of animals subjected to inhalation of low oxygen revealed an increase in the lactic acid proportional to the degree of the anoxia. It has also been shown that the lactic acid in the postmortem brain tissue is markedly increased. The study of the amount of lactic acid in the brain is thus a measure of the normalcy of the oxidative processes in that tissue. The brain of the experimental animal subjected to cranial injury revealed normal values except in the areas of contusions and lacerations. At such sites the lactic acid was markedly increased, denoting evidence of the defective local oxidation of glucose.[10]

Specimens of brain tissue by this technic were obtained as early as fifteen seconds after impact, and it was found that the "concussive" phenomenon is accompanied by normal levels of lactic acid. The concept that anemia of the brain at the time of impact is the cause of the post-traumatic unconsciousness is well disproved.[11]

In the experimental animal the use of a high concentration of oxygen, and oxygen with 5 per cent carbon dioxide, for as long as forty-eight hours does not materially change the disturbed oxidative mechanisms in traumatic contusions. However, it was found that when the animal was maintained at anoxic levels of oxygen, the addition of carbon dioxide increased the percentage of oxygen saturation (unpublished data).

COMPOUND FRACTURES, GUNSHOT AND OTHER PENETRATING WOUNDS

The management of penetrating wounds and the mechanism of wounding by high velocity missiles have received considerable attention during World War II.[12] The "temporary" cavity about the passing bullet, so beautifully shown by Butler, Puckett, Harvey and McMillan [13] and Black, Burns and Zuckerman,[14] is the cause of the extensive pulping of brain tissue at considerable distances from the region of the track. With this picture clearly in mind, it is no wonder that the successful management of such cases depends on the thorough debridement of the pulped brain, removal of foreign particles and fragments of bone. The long tedious technic of osteoplastic flap has been replaced by a complete removal of the area of shattered bone with a rongeur. If at all possible, the dura is closed if necessary with the use of fascia lata, pericranium, or temporal fascia. In extensive loss of tissue, all effort is made to cover the exposed brain with transposed scalp. Exposures are made through long curved incisions in order to enable the complete excision of the wound of entrance. The management of the cases in which there is infection, particularly those with abscesses of the brain, has been simplified by the use of chemotherapy and penicillin and by a policy of a complete excision of the infected brain, foreign bodies and fragments of bone [12] (fig. 5).

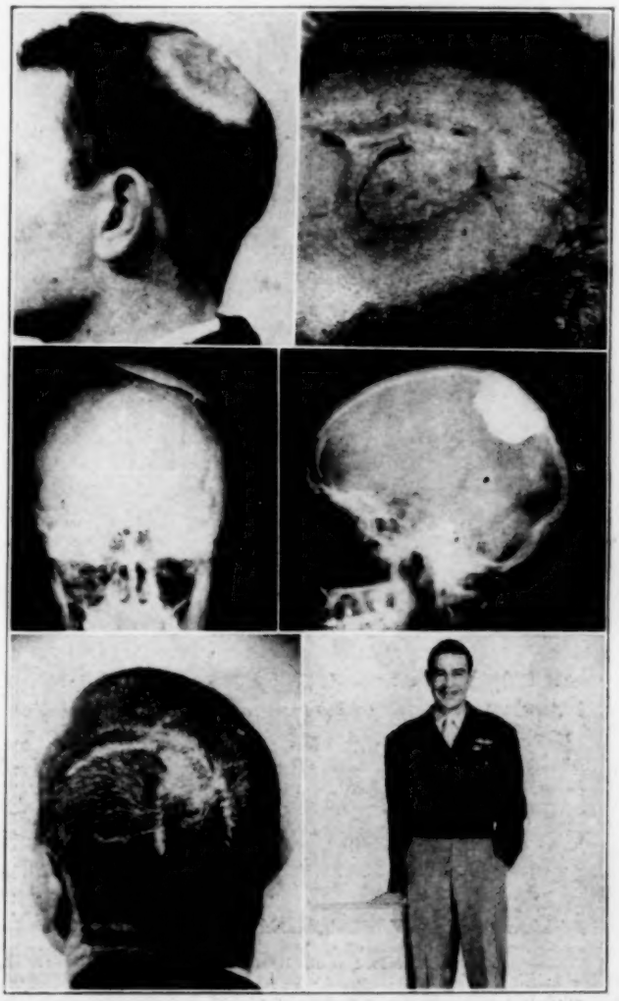

Fig. 5.—In the upper row are shown the posteroanterior and lateral views of an abscess of the brain due to penetrating wound by shell fragment treated by exteriorization, excision of abscess, shell fragments and necrotic brain tissue, followed by closure with fascia lata. Ten days later the fascia lata transplant was removed and a split thickness graft of skin was used. The middle row, following tantalum repair of skull defect. Lower row, the patient as he appears today.

In cases of civilian gunshot wounds of the brain, if the patient shows evidence of resuscitation by transfusions and other therapy, a complete and thorough debridement of his wound should be undertaken as soon as possible. But if, because of his poor condition, he is not operated on for two or more days, a delayed primary debridement may be safely accomplished. In a case of a gunshot wound of the right frontotemporal region, the debridement was done four days after

9. Stone, W. E.: J. Biol. Chem. **149**: 29, 1943.
10. Gurdjian, E. S.; Webster, J. E., and Stone, W. E.: Surg., Gynec. & Obst. **78**: 618, 1944.
11. Gurdjian, E. S., and Stone, W. E.: Federation Proc. **5**: 38, 1946.
12. Webster, J. E., and Gurdjian, E. S.: Internat. Abstr. Surg. **82**: 353, 1946.
13. Butler, E. G.; Puckett, W. O.; Harvey, E. N., and McMillan, J. H.: J. Neurosurg. **2**: 358, 1945.
14. Black, A. N.: Burns, B. D., and Zuckerman, S.: Brit. M. J. **2**: 872, 1941.

admission to the hospital. Almost all of the frontal lobe on the right side was removed, the dura was repaired with temporal fascia and the wound was closed in layers without draining. No complications arose and the wound healed by primary intention.

In the management of compound and simple depressed fractures in civilian life, some preoperative forethought in planning may minimize postoperative defects (fig. 6). In the absence of a torn dura, some of the fragments of bone may be used to advantage to obviate a later repair of the skull defect. The use of an inanimate transplant at the time of repair of a depressed fracture, as advocated by Gardner [15] may be undertaken with confidence except in

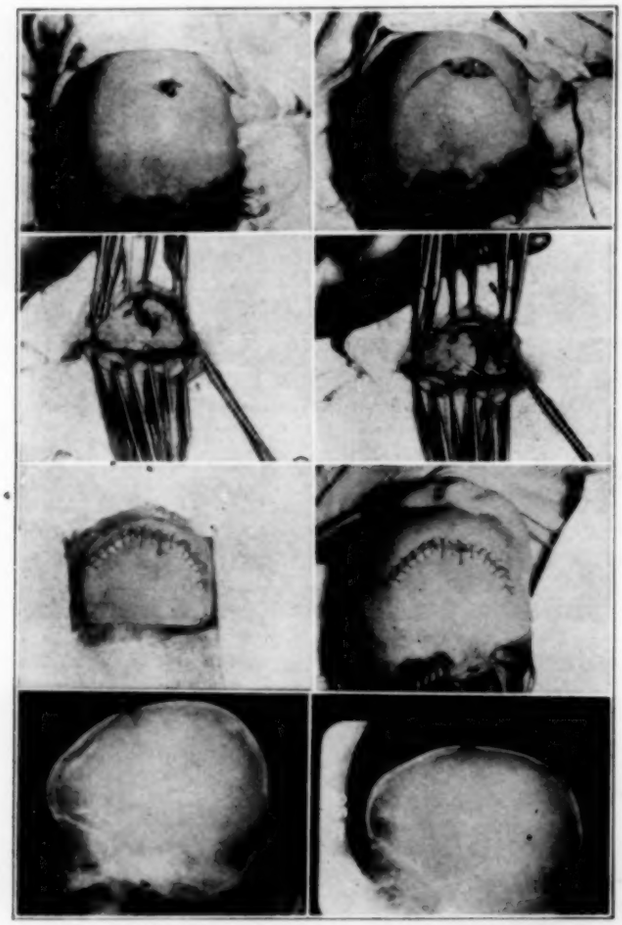

Fig. 6.—Compound depressed skull fracture of the midfrontal region at the hairline. The laceration was excised through a curved excision. The dura was found intact. The depression was elevated with little loss of bone tissue. Some preoperative forethought will obviate postoperative defects which might necessitate later repair.

cranio-orbital and frontal sinus wounds, where later repair of the defect is preferable, since the purpose of the primary operation is to obviate intracranial infection by preventing the formation of cranionasal fistulas.

In compound fractures, the initial wound may be excised in toto in the greatest majority of the cases by the use of long curved incisions (fig. 6). This is preferable to the usual method of excising only the edges of the wound.

The repair of skull defects has received considerable attention during World War II. Osteoperiosteal transplant from the outer table of the skull may be used for small defects and for the younger patient. Tantalum [16] and methyl methacrylate [17] have been proved to be sufficiently inert to make their use safe in the larger defects of the skull. The preparation of the transplant before operation for a satisfactory postoperative result is important, and has been suggested by several authors.[18]

15. Gardner, W. J.: U. S. Nav. M. Bull. **43**: 1100, 1944.

CONCLUSIONS

1. Head injury may be by direct or indirect violence. Compression, acceleration or deceleration ensue. In most injuries a combination of compression, acceleration and deceleration takes place.

2. The amount of absorbed energy by the head in an optimum period of time governs the severity of the physiologic effects, whether the head is fixed or free to move.

3. A blow may result in deformation of the skull, increase in intracranial pressure and mass movements of the brain. The latter is more frequent when the head is free to move.

4. Fracture from general deformation of the skull may result from an outbending following the initial inbending after impact. Depending on the shape and velocity of the injuring object, there may be a localized or a generalized deformation.

5. Most fractures, including depressed fractures, result from tension (tearing apart) rather than from compression.

6. The oxidative processes of the brain proceed in normal fashion after moderate and severe head injuries, except in areas of contusion and laceration of the brain. At such sites there is a defective oxidation of glucose. In the experimental animal the arteriovenous differences of oxygen, carbon dioxide and glucose are within the range of normal in the moderately and profoundly injured.

7. In civilian life many simple and compound depressions may be repaired in such a manner as to prevent defects of the skull.

8. When it is necessary to repair a skull defect, an osteoperiosteal transplant from the outer table of the skull may be used for small defects and in the young. Tantalum and methyl methacrylate may be used for the larger defects. In some cases the repair of the defect may be undertaken at the time of the primary operation.

9. In gunshot wounds of the brain, operative treatment is indicated as soon as the patient's condition permits. With the use of chemotherapeutic agents, penicillin and early debridement, intracranial infection develops in few cases.

ABSTRACT OF DISCUSSION

MAJOR EARL WALKER, Cushing General Hospital: One has noted both in civilian and military experience that patients who suffer minor wounds to the head will frequently lose consciousness, whereas other patients suffering severe compound wounds of the skull will have no period of unconsciousness whatsoever. The studies here presented enable us to understand this phenomenon. When a blow is struck to the head, two phenomena occur within the head: first, the deformation of the skull, with the subsequent waves traveling along the skull, and secondly, intracranial pressure waves traveling through the brain substance. Physicists state that compression alone does not cause alteration

16. Pudenz, R. H.: Repair of Cranial Defects with Tantalum, J. A. M. A. **121**: 478 (Feb. 13) 1943. Robertson, R. C. L.: J. Neurosurg. **1**: 227, 1944. Mayfield, F. H., and Levitch, L. A.: Am. J. Surg. **67**: 319, 1945. Hemberger, A. J.; Whitcomb, B. B., and Woodhall, B.: J. Neurosurg. **2**: 21, 1945.

17. Gurdjian, E. S.; Webster, J. E., and Brown, J. C.: Surgery **14**: 876, 1943. Elkins, C. W., and Cameron, J. E.: J. Neurosurg. **3**: 199, 1946.

18. Baker, G. S.: Proc. Staff Meet., Mayo Clin. **21**: 177, 1946. Footnote 17.

in nerve function. It is interaction of the forces produced by the intracranial pressure and the deformation of the skull which gives rise to the concussive phenomenon. The observations of Dr. Gurdjian do more than satisfy our scientific curiosity. He has shown us that in cranial concussion, there is no anoxia and there is little anoxia of any other portion of the brain unless contusion occurs. We know from other experiments that if anoxia develops in the blood cerebral edema will occur. So in the central nervous system, if there is any obstruction to the air passages, a pronounced cerebral edema develops, not due to the actual damage to the brain itself, but due to the generalized anoxia. It is therefore important, and a fact which is frequently overlooked, that the air passages be kept perfectly free of mucus and foreign bodies such as teeth, so that no such anoxia will develop.

DR. E. S. GURDJIAN, Detroit: Dr. Walker gave an excellent discussion on the mechanism of concussion. I think one other point that should be emphasized is that we are dealing with dynamic pressures, not static pressures. A hydrostatic pressure in an enclosed cavity such as the skull may cause no damage even if it were at high levels, but when dynamic pressure waves pass through the brain at speeds of $\frac{1}{1,500}$ second or less, shear and tear of tissue may take place in their path, and experimental and clinical work supports this concept.

WEIL'S DISEASE

Observations in Sixty-One Cases with Special Reference to the Use of Penicillin in Six Cases

H. M. PATTERSON, M.D.
Olaa, Hawaii, T. H.

A complete discussion of Weil's disease will not be attempted in this paper. In a previous paper [1] I gave a general report of observations in 37 cases of Weil's disease, and another paper [2] now ready for publication reports observations in a larger series of 61 cases. Only such general considerations as are necessary to bring out points in the management and treatment of this disease will be discussed in this presentation.

To the average physician the term Weil's disease suggests infectious (spirochetal) jaundice, but since nothing like all patients ill with this disease show jaundice the newer term leptospirosis is probably preferable. However, due to common usage the term Weil's disease will be used throughout this paper. This disease is probably not nearly as uncommon or as severe in the Western Hemisphere as the medical literature indicates. An editorial in THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION of Oct. 11, 1941 [3] stated that only 67 cases of this disease had been reported in the American literature, with different writers reporting a mortality rate from 5 per cent to 40 per cent or about 30 per cent in cases with jaundice. In 1941 Larson [4] reported 51 cases from various parts of the United States and Puerto Rico, some of which may have been included in the 67 cases mentioned previously. Lester and his co-workers [5] in 1942 reported 14 cases; in 1943 Bruno and his co-workers [6] reported 15 cases, and in 1944 Patterson [1] reported 37 cases. A number of reports of smaller groups of cases have been made since 1941, but probably the total number of cases reported in the American literature is less than 300. Since the literature is made up of reports of a few cases from widely scattered areas it would be expected that 1 death would cause the mortality rate to appear unduly high; likewise, the physician treating an occasional patient would possibly not always institute the treatment program most likely to result in recovery. The latter point in my belief is certainly true, since I consider that the best single step in the treatment of the patient severely ill with Weil's disease is to give whole blood transfusions from donors who have recovered from this disease. Such transfusions would not be generally available, since so few cases are on record in the average community. In the 61 cases on which this report is based there was not a single death. I do not believe that this lack of mortality is due to any lessened virulence of the Leptospira icterohemorrhagiae in this area, because I have seen a death from this disease in a neighboring district and have had reports of other deaths in the Territory of Hawaii. Likewise 6 of the patients in this series were desperately ill, apparently to a degree similar to that of the patient in the average case outlined in the literature. Probably with a few exceptions, only the relatively small number of exceedingly severe cases are being diagnosed as Weil's disease, leading to the conception that the disease is both severe and infrequent. It is considered that the treatment instituted in the 6 severely ill patients of this series saved the lives of some of them and that the treatment given to the remaining 55 patients prevented some of them from becoming severely ill. Undoubtedly, one reason why such a large series of cases of Weil's disease can be reported with no deaths is the fact that most of the patients were mildly ill. Only because of my increasing clinical experience with the disease were their cases ever correctly diagnosed. It is felt that nearly all instances of anicteric Weil's disease are being diagnosed as influenza or some other disease and that the illness of many of those patients who become mildly jaundiced is never correctly diagnosed because of the lack of laboratory facilities, because the proper laboratory tests are not performed or because the patients recover and return to work before the laboratory tests are completed or repeated. No attempt will be made in this paper to discuss the merits of the various laboratory procedures used in the diagnosis of Weil's disease. The procedure I consider most reliable is a rising titer shown in microscopic blood serum agglutination tests on specimens taken at weekly intervals during the illness and in the convalescent period. Such a test was positive in each of the 61 patients of this series in dilutions of at least 1:300; in all but 5 patients the agglutination tests were positive in dilutions of 1:1,000 or higher, and in all the patients on whom the tests were repeated either there was a rising titer or the titer was extremely high (1:10,000 or higher) in all tests. All the agglutination tests were performed by Dr. J. E. Alicata of the Parasitology Laboratory of the University of Hawaii.

The blood serum agglutination tests are performed on the first, eighth and fifteenth days after admission to the hospital. Often the patient has been discharged and must return for the tests on the fifteenth day after admission. The tests are negative on the first day, usually show no titer or a low titer on the eighth day

1. Patterson, H. M.: Weil's Disease: A Report of Thirty-Seven Cases, Hawaii M. J. **3**: 213-221 (May-June) 1944.
2. Patterson, H. M.: Weil's Disease: Observations Based on Sixty-One Cases, to be published.
3. Weil's Disease, editorial, J. A. M. A. **117**: 1266-1267 (Oct. 11) 1941.
4. Larson, C. L.: Weil's Disease: Report of Fifty-One Cases in Puerto Rico and and the United States, Pub. Health Rep. **56**: 1650-1656 (Aug. 15) 1941.
5. Lester, B. F.; Denison, G. A.; Posey, L. C., and Tale, G. M.: Weil's Disease: Clinical and Epidemiological Report of Fourteen Cases, South. M. J. **35**: 325-332 (April) 1942.
6. Bruno, F. E.; Wilen, C. J. W., and Snavely, J. R.: Spirochetal Jaundice: Report of Fifteen Cases, J. A. M. A. **123**: 519-524 (Oct. 30) 1943.

and show a high titer or an increasing titer on the fifteenth day. In leptospirosis due to L. icterohemorrhagiae these tests will always be positive with the procedure now being used in the Parasitology Laboratory of the University of Hawaii. If an infection should be due to other strains of the Leptospira, specific antigens must be used. As brought out in a general report [2] I consider that it is possible that occasional infections with other strains of the organism do occur in this area, but they are rare and not of practical importance in this report. With a laboratory test that is about 100 per cent efficient in the final diagnosis of Weil's disease, it is felt that this test should be used and relied on in preference to tests for Leptospira in the blood and urine or inoculation of laboratory animals with urine or blood or their concentrates. It is true that in the average infection in this area the patient is nearly well of his disease when the laboratory tests confirm the clinical diagnosis, or at least the patient has passed through the stage when early, adequate treatment will modify the disease. However, all other laboratory tests which are available, and which are less efficient, in a majority of cases are not positive until the golden period for institution of the proper treatment has passed, namely, the first few days of the illness. I believe and have tried to clearly report [7] that the diagnosis of Weil's disease should be made on the basis of the early clinical signs and symptoms and that treatment should be begun on the first day of admission to the hospital. If the clinician will have a highly suspicious frame of mind he can make this diagnosis on the first day. The average patient presents a picture of moderately severe influenza with decided conjunctival and scleral engorgement. If the clinician remembers just this much he will usually make the correct diagnosis. The ocular signs are the key to the diagnosis of Weil's disease in the early stage. In more detail, the average patient will have on admission generalized malaise, anorexia, headache (usually frontal), chills, generalized muscular tenderness and weakness (often severe in the calf muscles), a listless and toxic appearance and the classic ocular signs of decided conjunctivitis and scleritis. The eyes will appear soiled and will vary from pink to red with a cyanotic hue and with no discharge or secretions. In the average patient there will be no jaundice or bleeding or enlargement of the liver or spleen on admission. The temperature may be as high as 104.2 F. but will average 101 F., while on the average the pulse rate will be 91, the respiratory rate 22, the white blood cell count 11,704 with no significant differential change, the red blood cell count between 3.5 and 4 million and the hemoglobin 65 to 70 per cent, with the urine showing 1 plus albumin and no cells or casts. If this composite picture is kept in mind, with emphasis on the ocular signs and with attention being paid to the occupation of the patient, the diagnosis of Weil's disease can be made within a few hours after admission and treatment can be started at once. This point cannot be overemphasized. The final diagnosis is established by the blood serum agglutination tests on the fifteenth day.

Between Oct. 1, 1941 and April 16, 1946 61 patients with proved cases of Weil's disease were admitted to the Olaa Hospital at Olaa, Hawaii, T. H. All 61 patients came to the hospital for treatment of a clinical illness, and each presented signs and symptoms suggestive of Weil's disease, the diagnosis being confirmed in every case by microscopic blood serum agglutination tests with L. icterohemorrhagiae antigen. All the patients, except 1 who come from a bordering ranch, were residents of the Olaa district of the island of Hawaii, which has a population of 7,500. The entire treatment of the 61 patients was carried out in one hospital under my direction. The treatment regimen has evolved during the four and one-half years of this study, and it is not claimed that the final answer has yet been found.

TABLE 1.—*Length of Illness and Other Data, Broken Down According to Special Treatment Received; All Patients Received the Same General Treatment*

Special Treatment	No. of Patients	Day in Hospital Temperature First Dropped to Normal (Average)	Day in Hospital Temperature Remained Normal (Average)	Days in Hospital (Average)	Days from First Symptom to Discharge from Hospital (Average)
Entire series (as below)	61	4.2	5.5	12.08	15.28
No special treatment	44	3.8	6.0	9.00	12.16
Sulfathiazole	7	7.8	9.7	19.30	21.50
Sulfathiazole plus whole blood from donors who had not had Weil's disease previously	1	2.0	22.0	38.00	40.00
Sulfadiazine plus whole blood from donor who had previously had Weil's disease	1	4.0	31.0	35.00	36.00
Whole blood from donors who had previously had Weil's disease	2	7.5	7.5	18.00	20.00
Penicillin	6	4.17	5.5	9.00	12.28

TREATMENT WITH SULFONAMIDE COMPOUNDS

The first 3 patients of the series were so ill that it was decided to treat them with sulfathiazole, 1 Gm. every four hours, this being continued for from three to ten days. Thereafter, courses of sulfathiazole, 0.5 to 1 Gm. every four to six hours according to the weight of the patient, were given to 5 other patients for from three to ten days and sulfadiazine, 1 Gm. every four hours for two days, was given to 1 patient. All these patients grew progressively worse while receiving this medicament, and thereafter it was not used again. See table 1 for comparable data with other special treatment programs. It is only accurate to state that of the 9 patients who received a sulfonamide compound 6 were decidedly ill, and a longer hospital stay would normally be expected for them than for the average patient in this series. However, when the sulfonamide therapy was stopped in 3 of the more seriously ill patients and blood transfusions were given improvement was so prompt that it was felt that medication with the sulfonamide drugs had been of no value.

TREATMENT WITH BLOOD TRANSFUSIONS

After sulfathiazole had been discontinued in the second patient of the series it was decided to give him transfusions of whole blood. No donors who had been

7. Patterson, footnotes 1 and 2.

proved to have had Weil's disease were available, so other donors were used. Improvement was definite but slow after the first transfusion of 550 cc. of citrated blood, so two other such transfusions were given with slow but certain improvement. At this point it was decided that whole blood from donors who had had Weil's disease should be the best treatment in future severely ill patients. The early cases were grouped closely together and the patients were all so ill that it was felt too hazardous to complicate their convalescence by using them as donors. Specimens of blood from all patients were typed and were Kahn tested. Patient 28 of the series was desperately ill and lost 30 pounds (13.6 Kg.) during his illness. On the ninth hospital day he was given 500 cc. of citrated blood from a donor who had had Weil's disease fourteen months before and whose blood titer had been 1:30,000 on his recovery at that time; the titer was not determined again at the time when blood was taken. Patient 28 was bleeding from the mouth and nose, was vomiting all food and liquids and was desperately ill when the blood transfusion was given. Thereafter he did not vomit, had no bleeding, became alert and proceeded to get well. The change was prompt and dramatic.

Patient 47 was desperately ill on the seventh day in the hospital and insisted that she was going to die. She was extremely toxic, her jaundice was deepening and she was vomiting everything taken orally. A blood transfusion was started, using as the donor a man who had had Weil's disease two months before and whose blood titer was 1:30,000 at the time of the transfusion. After receiving 300 cc. of citrated blood the patient had a severe chill, which lasted thirty minutes. Thereafter she felt better and ate her supper without vomiting. The following day she was given the remaining 250 cc. of the transfusion which had been started the preceding day. She had no chill and was laughing before the transfusion was finished; her temperature dropped to normal the same day and did not rise again. She ate heartily thereafter and said she felt that a miracle had happened to her. She went home six days later.

Patient 50 was deeply jaundiced on admission and was weak and listless. He vomited everything taken by mouth. His liver was enlarged, and he was spitting blood. These symptoms progressed, and on the third day he was given 550 cc. of citrated blood from his brother, who had had Weil's disease two years and one month previously and whose blood titer was only 1:100 at the time of the transfusion. The patient had a slight chill which lasted ten minutes immediately after the transfusion, and he vomited. Thereafter he felt better; his temperature dropped to normal within twenty-four hours and did not rise again. However, he was not discharged until eighteen days later because of the deep jaundice, which slowly faded, and because of the enlarged liver. He had no bleeding from the mouth after the transfusion.

Much is written of developing a serum or obtaining serum from biological houses for the treatment of Weil's disease. Walch-Sorgdrager,[8] Taylor and Goyle [9] and nearly every textbook discussing this disease emphasize this type of treatment. Patients with the disease need not only serum but red blood cells and all the constituents of whole blood. In this entire series the average lowest red blood cell count was 3,755,000 and the average lowest hemoglobin was 66 per cent. In those patients who were jaundiced the average lowest red blood cell count was 3,331,000 and the average lowest hemoglobin was 64.8 per cent. All the patients of this series have had their blood typed and Kahn tested and in my opinion constitute the best source of transfusion for the treatment of future severely ill patients with Weil's disease in this area. Several of them have given blood to patients with this disease in neighboring districts, with prompt improvement of the recipients. See table 1 for comparable data with other specific treatments. In consulting this table it should be kept in mind that the patients who received blood transfusions as well as those who received the sulfonamide compounds were the most severely ill patients of the series, in whom a longer hospital confinement would be expected.

TABLE 2.—*Treatment of Six Patients with Penicillin*

Patient	Days Ill Before Admission	Jaundice	Bleeding Before Admission	Bleeding in Hospital	Penicillin Sodium Intramuscularly	Temperature (F.) on Admission	Hosp. Day Temp. First Reached Normal	Hosp. Day Temp. Remained Normal	Days in Hospital
56	6	Scleral, mild	Epistaxis	.....	15,000 U. every 3 hrs. for 53 doses	103.6	7	7	9
57	1	.....	.....	Epistaxis	15,000 U. every 3 hrs. for 40 doses	104.0	7	7	10
58	3	.....	.....	Hemoptysis	15,000 U. every 3 hrs. for 48 doses	102.0	6	6	11
59	5	Scleral, mild	.....	Epistaxis	15,000 U. every 3 hrs. for 32 doses	99.0	2	2	6
60	1	.....	.....	.....	15,000 U. every 4 hrs. for 36 doses	104.0	2	6	9
61	4	.....	.....	.....	15,000 U. every 3 hrs. for 48 doses	104.0	1	5	9

PENICILLIN THERAPY

In February 1944 Heilman and Herrell of the Mayo Clinic [10] reported good results with penicillin in the treatment of experimental infections of leptospirosis due to L. icterohemorrhagiae in guinea pigs and suggested that this drug should be effective in the treatment of such infections in man. Augustine and his co-workers [11] in September 1944 after experimental use of penicillin in laboratory animals concluded that penicillin has a suppressive effect on L. icterohemorrhagiae in a highly susceptible host when given before the appear-

8. Walch-Sorgdrager, B.: Leptospirosis, Bull. Health Organ., League of Nations **8**: 143-386 ,1939.

9. Taylor, J., and Goyle, A. N.: Leptospirosis in the Andamans, Indian J. M. Research (Memoir no. 20) March 1931, pp. 1-190.

10. Heilman, F. R., and Herrell, W. E.: Penicillin in the Treatment of Experimental Leptospirosis Icterohaemorrhagica (Weil's Disease), Proc. Staff Meet., Mayo Clin. **19**: 89-99 (Feb. 23) 1944.

11. Augustine, D. L.; Weinman, D., and McAllister, J.: Penicillin Sodium Therapy in Experimental Weil's Disease, New England J. Med. **231**: 358-359 (Sept. 7) 1944.

ance of clinical symptoms. These workers did not expect it to be effective after the development of clinical symptoms. In March 1945 Larson[12] of the United States Public Health Service in a similar study with young Swiss mice found that penicillin had a definite therapeutic effect on the course of the disease when administered in the first forty-eight hours, but thereafter its effectiveness was materially decreased. He also treated a comparable group of mice with immune serum and found it equally as effective as penicillin.

In line with these reports I have treated the last 6 patients of this series with penicillin, beginning on the first day of the illness in each case. Sodium penicillin dissolved in isotonic solution of sodium chloride was used intramuscularly (table 2). In none of these patients treated with penicillin were the liver or spleen enlarged. The temperature dropped by crisis in patients 58 and 59 and by lysis in the other 4. These 6 patients presented a clinical picture on admission corresponding to the average for the entire series. With 2 of these patients showing mild jaundice and with 1 showing epistaxis before admission and with 2 showing epistaxis after admission and 1 showing hemoptysis after admission, it might have been expected that 1 or 2 of these patients would have become much more severely ill than they did. It is definitely felt that penicillin therapy modified the course of the disease. Though table 1 shows that patients in the penicillin-treated group compare closely to those in the group receiving no specific treatment it is felt that on the average the latter were not as ill as the former.

GENERAL TREATMENT

Patients with Weil's disease should have good care by the nursing staff and close observation by the physician in charge. These patients need care similar to that given to patients with typhoid fever. The fluid intake must be watched closely and parenteral fluids given as needed; in this series parenteral fluids were required in 12 per cent of the cases. Attention to oral hygiene is necessary. The patient must be encouraged and reassured. Analgesics for the relief of headaches and generalized aches and pains should be used freely and in adequate doses, being required in 65 per cent of the cases in this series. Acetylsalicylic acid in 10 grain (0.65 Gm.) doses provides satisfactory relief of these symptoms. Twenty-two patients in this series required measures to combat constipation, enemas usually being used. Epistaxis was present in 33 per cent of the cases and required treatment ranging from ice compresses to postnasal packs. Vitamin K is given to all patients, 2 mg. by mouth daily, and if jaundice or bleeding occur 10 mg. is given daily hypodermically. Because of the almost constant anemia ferrous sulfate and extracts of liver are given by mouth. Capsules containing all of the vitamins are given in large dosages because of the generally poor intake of food and the great loss of body weight, which averaged 10.8 pounds (4.9 Kg.) in this series. Likewise, these patients need a liver-protecting and tissue-building diet, and in this series a high caloric, high carbohydrate, high protein diet has been used. Because the kidneys are potentially damaged and because the Leptospira does poorly in an acid urine, tablets of methenamine and sodium biphosphate, which contain 10 grains (0.6 Gm.) of each, are given every eight hours during the hospital stay and for one week after discharge. Other signs and symptoms which occur less frequently are treated as they arise. In general no treatment is required for the eyes during the acute phase of the illness, but attention should be called to the frequent ocular complications, principally iritis, iridocyclitis and uveitis, which require treatment as they develop from one month to one year or more after the acute illness. In from 20 to 25 per cent of the patients in this series such complications have developed.

PREVENTIVE MEASURES

In this series 51 of the 61 patients were cane cutters and most of the others were exposed to the hazards of the cane cutter. The answer to the mode of entry of the organism has not been found, but all such workers should drink only pure water, should protect their extremities and faces as much as possible from abrasions, should wash their hands thoroughly before eating lunch in the fields and should avoid rubbing the eyes or wiping sweat from the face with soiled hands. Measures that will destroy the rat and mongoose population of this area will probably cause the disappearance of Weil's disease. The development of a vaccine to be given to field workers in this area seems to be indicated. After release from the hospital the convalescent patient should be given methenamine and sodium diphosphate in large doses in order to help destroy the Leptospira, which are shed in the urine for many weeks.

SUMMARY

1. General observations of the management of 61 cases of Weil's disease are reported.

2. Sulfathiazole was used in 8 patients and sulfadiazine in 1 patient with no apparent benefit.

3. Three whole blood transfusions from donors who had not previously had Weil's disease were given to 1 patient, who obtained slow but progressive improvement after each transfusion.

4. Single transfusions of whole blood were given to 2 patients and two smaller transfusions to 1 patient, using donors who had previously had Weil's disease, with prompt and certain improvement in each instance.

5. Penicillin was used from the day of admission in the last 6 consecutive cases with apparently excellent results. None of these patients became severely ill, and their periods of hospitalization compare closely to those of the mildly ill patients of the series who received no special treatment.

6. The general measures for hastening recovery are outlined, and preventive measures are discussed.

CONCLUSIONS

1. All patients with Weil's disease are potentially dangerously ill and require the best of nursing and medical care of the type such as given to the patient with typhoid fever.

2. The use of transfusions of whole blood from donors who have had Weil's disease should be the sheet anchor in the treatment of the severely ill patient.

3. The administration of penicillin from the first day of hospitalization is recommended for all patients with Weil's disease.

4. The sulfonamide compounds do not appear to be effective against Weil's disease.

12. Larson, C. L.: A Comparison of the Effect of Penicillin and Immune Serum in the Treatment of Experimental Leptospirosis in Young White Mice and in Howsters, Pub. Health Rep. **60**: 319-323 (March 23) 1945.

MYOMETRIAL PHYSIOLOGY AND ITS RELATION TO PELVIC PAIN

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Many factors contribute to the presence of pelvic pain during menstruation. Attention in the present paper will be devoted primarily to the myometrium, though the person as a unit must always be considered. The present knowledge concerning human nonpregnant uterine contractions is based on extensive investigations on experimental animals supplemented by careful clinical observations and uterine pressure records from control, pregnant and dysmenorrheal patients. Intrauterine pressure records from the nonpregnant human uterine were first obtained by Moir,[1] who observed during menses that contractions occurred every two minutes and developed pressures as high as 100 mm. of mercury. No pain but a feeling of heaviness and discomfort occurred when uterine pressures of 120 mm. of mercury were present. In patients with severe dysmenorrhea, pressures of 150 mm. of mercury were recorded and vascular pulsations disappeared at 120 mm. of mercury. Bickers[2] showed that high uterine tone is associated with primary dysmenorrhea and that reduction in tone is associated with relief from distress.

The literature contains many references to neurogenic and myogenic types of dysmenorrhea; and it is generally accepted that acetylcholine and histamine are agents which could produce these types. Yet drugs such as "trasentin" (the hydrochloride of diphenylacetylaminoethanol) and "pavatrine" (beta-diethylaminoethyl fluorine-9-carboxylate hydrochloride), which are excellent spasmolytics to animal tissues exposed to acetylcholine, histamine or barium,[3] often fail to relieve the distress of dysmenorrheal patients. Why? As the first step in answering this question, it seemed advisable to determine whether acetylcholine and histamine would produce uterine contractions and pain in human beings.

Methods.—Uterine activity has been investigated with the aid of pressure tracings and electrouterograms. Balloons fastened to short sections of ureteral catheters size 4 were placed in the uterus and cervix and then connected to optical manometers of high frequency.[4] The recording systems and balloons were filled with fluid so that transmission of uterine pressure changes to the recording device was accomplished by displacement of less than 0.01 cc. of fluid. Accurate tracings could be obtained with only 0.1 cc. in the balloon, though usually 0.2 to 0.5 cc. was placed in the cervical balloons and 0.5 to 1 cc. in the uterine balloons. Advantages which this method offered included: (1) the use of exceedingly small balloons, (2) a minimal amount of foreign object reaction within the uterus, (3) opportunity to use multiple balloons and (4) ability to obtain true zero pressures and to appreciate the amount of tone present.

Previous efforts[5] to evaluate uterine activity by means of electrouterograms have been of limited value. In the present studies an electrocardiograph of the Einthoven type was adapted to move the photosensitive paper at slow speeds. Silver chloride-coated silver electrodes were placed either in the uterus and cervix or on the anterior and posterior lips of the cervix or in various positions on the skin of the patient. Clinically useful electrouterograms could not be obtained with extrauterine electrodes[6] but were obtained from electrodes inserted into the uterus and cervix (fig. 1, tracings 1 to 4). Interpretation of the electrouterograms from 28 patients frequently has supplied confirmatory evidence for the pressure records, but additional investigations will be required before full interpretation can be firmly established.

Production of Dysmenorrhea.—At various times in the menstrual cycle, especially during menses, acetylcholine and histamine have been injected intravenously into 6 control patients and 6 dysmenorrheal patients in amounts which produced definite discomfort, such as flushing, headache, intestinal contractions and tachycardia. Even though some injections were continued over a ten minute period none of these patients experienced the typical pain or distress associated with her menses or showed appreciable change in uterine activity (fig. 1, tracings 1 and 2).

Next in our attempt to intensify the symptoms of the dysmenorrheal patients and to produce them in the control patients, posterior pituitary preparations were injected intravenously. "Pitocin" (N. N. R.), even in doses which caused definite uterine activity (3, fig. 1), did not bring about any distress. In fact many of the patients felt better. "Pitressin" (N. N. R.), even in doses which supposedly had much less oxytocic activity than that present in the dose of "pitocin" given to the same patient, produced decided distress, definite uterine cramps and pains, increased amplitude and frequency of contractions and elevation of the uterine tone (4, fig. 1). There were similar observations in all 25 patients receiving "pitressin." In fact this preparation intensified or produced all the symptoms which that particular patient had experienced during her troublesome menstrual periods.

These observations suggest that effective uterine antispasmodics should have definite anti-"pitressin" activity toward the uterus. Drugs such as "trasentin" and "pavatrine" do not possess much anti-"pitressin" activity.[6] This may account for the disappointing clinical results with these drugs after preliminary pharmacologic studies seemed to offer so much.

The "pitressin" and "pitocin" used in these investigations were purchased on the open market.

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2. Bickers, W.: Uterine Contractions in Dysmenorrhea, Am. J. Obst. & Gynec. **42:** 1023-1030, 1941.

3. Samaan, K., and Saad, K.: An Experimental Study of Diphenylacetyldiethylaminoethanol Ester Hydrochloride, Quart J. Pharm. & Pharmacol. **11:** 679-691, 1938. Lehmann, G., and Knoefel, P. K.: Spasmolytic and Local Anesthetic Action of Some Derivatives of Fluorene-Carboxylic Acid and Related Compounds, J. Pharmacol. & Exper. Therap. **74:** 274-283, 1942.

4. Hamilton, W. F.; Woodbury, R. A., and Harper, H. T., Jr.: Physiologic Relationships Between Intrathoracic, Intraspinal and Arterial Pressures, J. A. M. A. **107:** 853-856 (Sept. 12) 1936.

5. Hasama, B.: Registration of Normal Action Current from Uterus. Arch. f. exper. Path. u. Pharmakol. **153:** 129-139, 1930. Falk, H. C., and Nahon, R.: Electrouterography, Am. J. Obst. & Gynec. **30:** 403-410, 1935. Jacobson, E.; Lackner, J. E., and Sinykin, M. B.: Electrical and Mechanical Activity of the Human Non-Pregnant Uterus, ibid. **38:** 1008-1020, 1939.

6. Unpublished observations by this laboratory.

Comparison of 3, figure 1, with 4, figure 1, shows that some patients have had greater uterine activity after "pitressin" than after "pitocin" even though smaller doses of the former were injected. This confirms the observation of Moir.[1] Apparently bioassay methods for these preparations are unsatisfactory, since in these patients "pitressin" excited the uterus more than "pitocin" did. The fact that the onset of the stimulative action of the former was so rapid constitutes strong evidence that its uterine action was direct and not dependent on ischemia secondary to vasoconstriction.

Some dysmenorrheal patients experienced temporary relief after urination. It seemed reasonable that the relief might be caused by the partial emptying of the uterine contents during micturation. This suggested that the stretching of the uterus by the accumulation of clots and menstrual fluid and débris contributed to the distress of the patient.

The influence of stretching the uterus by distention of the balloon was investigated. Dysmenorrheal patients as a group tolerated less stretch than did control patients. Dysmenorrheal nulliparous patients tolerated from 0.4 to 5 cc. with an average of 3.5 cc., while control nulliparas tolerated from 3 to 7 cc. with an average of 5 cc. Factors which may have contributed to this low tolerance in dysmenorrheal patients included underdevelopment of the uterus, lowered pain threshold, greater uterine muscle tone and greater edema and congestion in the uterus. Parous patients as a group tolerated greater distention of the balloon. This may explain the disappearance of dysmenorrhea with the first pregnancy in some patients.

Pressure Records of Control and Dysmenorrheal Patients.—The balloons imitated clots and stretched the uterus, which caused increased uterine activity. No sharp line of demarcation could be observed between the uterine tracings of control and dysmenorrheal patients. However, definite differences were present. During menses in control patients contractions occurred once every 1.5 to 3 minutes and developed maximal pressures of 60 to 150 mm. of mercury. Between contractions pressures of 5 to 15 mm. were present, indicating good relaxation. The uniformity and regularity of the contractions as well as the good relaxation between contractions indicated that the uterus was contracting as a unit. It is suggested that these should be called well coordinated contractions. In 5, figure 2, tracings from a control patient show coordinated contractions. The phenomenon of summation and of a relative refractory period[7] are illustrated. In dysmenorrheal patients the contractions were the coordinated type with extremely high pressures (6, fig. 2), the poorly coordinated type with high pressures (7, fig. 2) or the uncoordinated type with low pressures (8, fig. 2). In the coordinated, extremely high pressure type, the uterine musculature contracted as a unit and pressures of 140 to 340 mm. of mercury were encountered. Over a thirty minute period in 1 patient the pressures at the height of contractions averaged 230 mm. of mercury. These pressures greatly exceeded both those in the control patients and those of 40 to 90 mm. of mercury which are the limits of the maximal pressures developed by the uterus during parturition.[8] There is no doubt whatsoever that the pain of dysmenorrhea is real. It is not just psychic in such patients. The pressures between contractions were from 15 to 40 mm. of mercury in some patients and from 25 to 100 mm. in others. These uterine tone values were higher than the 5 to 15 mm. pressures of the control patients.

Uterine pressure pulsations corresponding in rate to the heart beat were observed in many patients. These pulsations frequently were reduced in amplitude or disappeared while the uterine pressure was elevated, but often they did not disappear even when the uterine pressure reached values much higher than the arterial pressure. Apparently, in some if not all patients these pulsations were transmitted directly from the throbbing abdominal aorta and did not represent pulsations of uterine blood vessels.

Contractions of short duration, which were generally not synchronous with contractions in surrounding portions of the uterus, have been named uncoordinated contractions. They frequently were superimposed on a background of coordinated contrac-

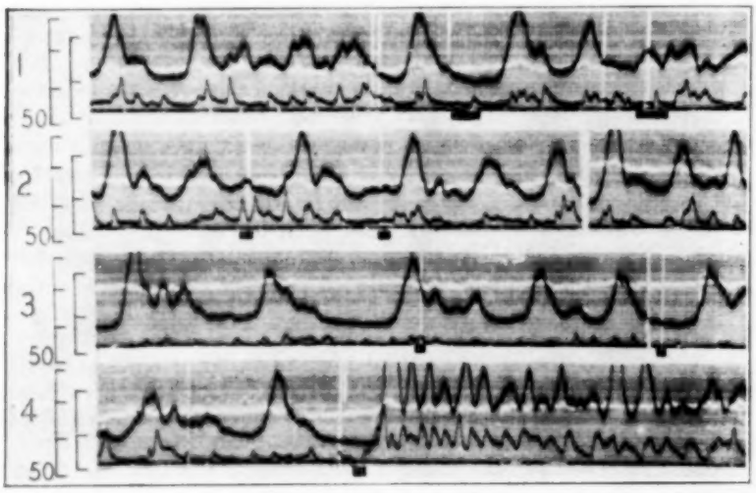

Fig. 1 (tracings 1 to 4).—Pressure records and electrouterograms: From above downward, tracings represent uterine pressure, electrouterogram, cervical pressure and base line interrupted at one minute intervals. Pressure scale: left-uterine 50, 100, 150, 200 mm. of mercury and right-cervical 0, 50, 100 mm. of mercury. These tracings, taken on the first day of menses when cervical biopsy indicated that ovulation had occurred, were secured from a patient who usually had slight but definite distress with her menses. Pronounced distress, but well within limits of tolerance, was induced by placing 3 cc. of fluid in the uterine balloon and 0.5 cc. in the cervical balloon. Note that: (*a*) the uterine pressures are high (induced by placing 3 cc. of fluid in the uterine balloon); (*b*) the cervical and uterine pressures are not equal and the contractions may or may not occur at the same time; (*c*) the time durations of the cervical contractions are much shorter than those of the uterine contractions, and (*d*) electrical potential changes occur during periods of uterine activity.

1. At each signal 9 mg. of acetylcholine was injected intravenously in 1.8 cc. of saline solution. Both injections, especially the second which was more rapid, caused a flush, a smothering sensation in chest and cardiac acceleration from 70 to 90 and 110 per minute. The patient noticed no change in uterine activity or in abdominal distress.

2. At each signal 0.005 mg. histamine as histamine phosphate was injected intravenously. The patient noticed no change in uterine activity or in her abdominal distress, though she felt some fulness and throbbing in her head.

3. At each signal 1 unit of "pitocin" was injected intravenously. The patient felt a slight transient sense of warmth but did not experience abdominal distress.

4. At the signal, 1 unit of "pitressin" was injected intravenously. This represents less than 0.05 unit of oxytocic activity and supposedly less than one twentieth of the oxytocic substance injected while recording tracings in 3, figure 1. The patient complained of abdominal cramps and backache just like she had with her "bad monthly periods." After about ten minutes she felt about the same as before this last injection. Note the decided increase in uterine activity which starts within 60 seconds of the injection. Most of this time is required for circulation of the drug from the cubital vein to the uterus. The fact that the onset of action is so rapid serves as strong evidence that the uterine stimulative action of this drug is direct and not secondary to uterine vasoconstriction.

7. Rudolph, L., and Ivy, A. C.: Coordination of Uterus During Labor, Am. J. Obst. & Gynec. **21**: 65-82, 1931.

tions and at intervals they temporarily replaced them (fig. 3). When recordings were obtained for several hours poorly coordinated or uncoordinated contractions would become superimposed on the background of coordinated contraction in the control patients and would dominate the tracings of dysmenorrheal patients

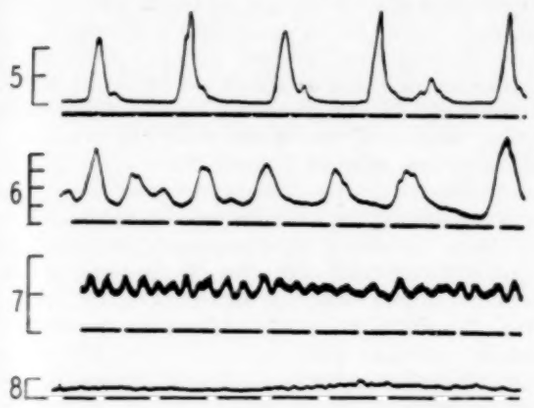

Fig. 2 (tracings 5 to 8).—Uterine pressure records from control and dysmenorrheal patients obtained on first day of menses: The base line was interrupted at one minute intervals. The pressure scale is in units of 50 mm. of mercury.

5. Uterine contractions from control patients are coordinated and good relaxation is present between contractions. The low tone between contractions and short periods of elevated pressure indicate that the balloon was in the lower uterine segment. A few accessory contractions occur. When these are present at the height of a contraction they elevate the pressure to abnormally high pressures. Only slight discomfort and no distress is present during these contractions.

6, 7 and 8. Tracings from 3 different dysmenorrheal patients: In tracing 6 poorly coordinated contractions are superimposed on coordinated contractions. Pressures are high during and between contractions. In tracing 7 poorly coordinated contractions dominate, indicating that incomplete tetany is present. The uterine tone is high and contractions fail to develop high pressure. However, the patient experienced much distress. In tracing 8 the pressures are not high, yet the patient was experiencing severe pain while this tracing was being recorded.

who originally showed just partial disorganization. Occasionally further distention of the balloon would bring about a temporary return of coordinated contractions.

The pressure at which patients complained of pain varied a great deal. When contractions were organized most patients complained of discomfort before pressures of 100 mm. of mercury were reached and of distress with pressures of 180 mm. of mercury. When contractions were disorganized and relaxation was inadequate, incomplete tetany (7, fig. 2) or complete tetany (8, fig. 2) developed. Under these conditions pain was present at pressures of 40 to 70 mm. of mercury. Extremely severe pain occurred while the tracing shown in 8, figure 2, was being recorded although pressures of only 20 to 30 mm. of mercury were present. It seemed advisable to determine whether such low pressures might be present in one part of the uterus while higher pressures were present at another level and were causing the pain.

Intrauterine Pressures in Different Parts of the Uterus.—Records were obtained simultaneously from three balloons inserted at different levels into the uterus and from one balloon inserted into the cervix. These tracings showed that the uterine pressure curves were different at various levels within the uterus both during coordinated contractions (fig. 4) and during uncoordinated contractions (fig. 5). Pain seemed to be present during uncoordinated contractions when pressures above 40 to 70 mm. of mercury occurred at any level within the uterus. As in the monkey,[9] uterine contractions are not peristaltic waves, in that no wave of relaxation precedes the contraction wave (fig. 4). Contractions may start in a region close to the juncture with the tubes,[9] yet the increase in pressure occurs first in the lower uterine segment and body of the uterus, then subsequently in the dome of the fundus. Downward from the dome of the fundus, the uterine muscles progressively contract faster and relax more completely. The musculature in the dome is slow to contract and even slower to relax (fig. 4). In menstruation as in parturition retraction of the lower uterine segment tends to occur. On the assumption that uterine contractions in labor and those in menstruation are similar, the records in figure 4 show that retraction of the uterus in parturition can be explained by the shorter contractions and the earlier and more complete relaxation of the muscles of the lower uterine segment as compared with those of the upper uterine segment.

The fact that different pressures exist in different portions of the uterus during menses allows coordinated contractions to propel clots and menstrual débris downward and to push arterial blood into the high pressure area of the dome of the uterus. The mechanisms by which these are accomplished and the influence of disorganized contractions on them will be described. When coordinated contractions occur during menses, relaxation brings about a large pressure gradient from the dome to the body of the uterus and from the body to the lower uterine segment (fig. 4, curves *A-B* and *B-C*). This means that débris is pushed downward within the uterus during the relaxation phase and not during the contraction phase. However, during the contraction phase there is a large pressure gradient between the lower uterine segment and the cervix, which moves débris into the cervix and thence into the vagina (fig. 4, curves *C-D* and *D*).

In patients with coordinated contractions, relaxation occurs between contractions and the strong uterine activity, though painful, will push débris and clots down and out of the uterus in the manner previously described. Contractions then may become reduced in intensity, and relief is obtained. In patients with disorganized contractions, relaxation between contractions

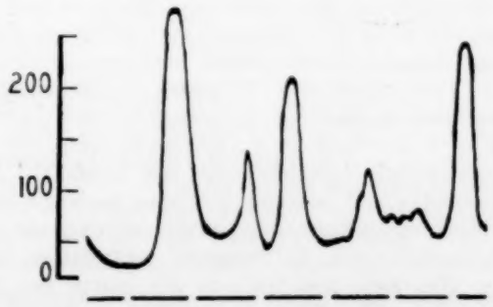

Fig. 3.—Uterine pressure record from patient with severe dysmenorrhea: This tracing is interesting in that the first contraction occurs after a period of adequate relaxation. Note the high pressure developed during this contraction. Severe distress was present during this contraction after the pressure was above 175 mm. of mercury and until it had decreased to 75 to 100 mm. of mercury. A short period of incomplete tetany at the right shows that poorly coordinated contractions were superimposed and replacing the coordinated contractions. Distress present during the period of incomplete tetany was severe but less so than that at the height of the first contraction.

is inadequate. Strong contractions develop sufficient pressure to clear the lower uterine segment but fail to

8. Woodbury, R. A.; Hamilton, W. F., and Torpin, R.: Relationship Between Abdominal, Uterine and Arterial Pressure During Labor, Am. J. Physiol. **121**: 640-649, 1938.

9. Ivy, A. C.; Hartman, C. G., and Koff, A.: The Contractions of Monkey Uterus at Term, Am. J. Obst. & Gynec. **22**: 388-399, 1931.

produce sufficient pressure to remove débris adequately from the body and dome of the fundus (fig. 5 and right half of fig. 4). Contractions continue, and dysmenorrhea of the type illustrated in 6, fig. 2, develops. This may continue, or the débris may be moved and relief obtained, yet in many of these patients there soon develop definitely disorganized contractions like those illustrated in 7, figure 2, or 8, figure 2.

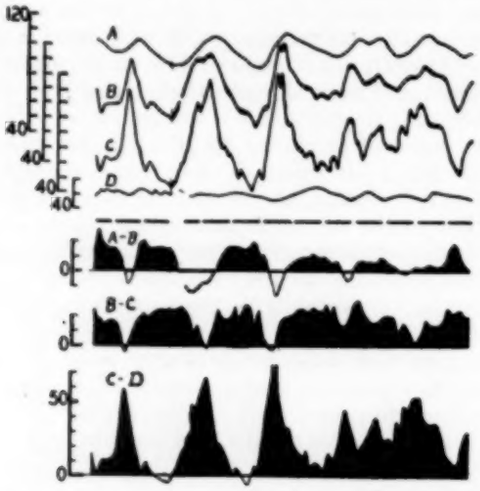

Fig. 4.—Pressure records were obtained simultaneously from four balloons placed within (*A*), the dome of the fundus, (*B*) the body of the uterus; (*C*) the lower uterine segment, and (*D*), the cervix. The curves were replotted to the same pressure scale, with each curve lifted the amount indicated at the left in order to prevent superimposition of lines. The pressure increases in the lower uterine segment and lower portion of body of uterus start earlier, occur more rapidly and are shorter in duration than those in the dome of the fundus. Curves $A - B$, $B - C$ and $C - D$ are differential pressures which show the pressure gradients between the dome and body of the fundus ($A - B$), between the body of the fundus and the lower uterine segment ($B - C$) and between the lower uterine segment and cervix ($C - D$). These differential pressures are the pressures which influence the passage of clots and débris. The coordinated contractions at the left provide waves of propulsion even though the uterine tone is high. The poorly coordinated contractions at the right fail to supply a wave of propulsion. Note also that during the height of contractions the pressure in the dome of the fundus is lower than that in the body of the fundus. During this time the pressure gradient is reversed. (See negative pressure values on tracing $A - B$). To a slight extent this would interfere with outward propulsion of uterine content, but it facilitates blood flow from vessels in the body of fundus to vessels in the dome.

An interesting record was obtained in figure 5; the pain of the patient was intense and the contractions were disorganized in type. Uterine tetany was present and pressures in the dome and body of the uterus were low, while pressures in the region of the third balloon, located in the lower uterine segment, were high. This might well be called a constriction ring during menstruation or a spasm of the isthmus, or it could result from an exceptionally long cervix. It would definitely interfere with the downward flow of débris and could account for this patient's distress. The injection of "pitressin" produced extreme distress and caused intense disorganized contractions of muscles in the dome and body of the uterus but did not further elevate the high pressure in the lower uterine segment. The pressures in the dome and body of the fundus approached but still did not reach those in the lower uterine segment. Apparently the musculature of the isthmus was already near maximal disorganization and maximal contractions, while the muscles in the dome and body had developed some inertia secondary to fatigue and insufficient blood supply.

During coordinated contractions the slow and incomplete relaxation in the dome should interfere with the blood supply to this area because the pressure remains high in this area. In 1 patient pressure in the dome remained over 260 mm. of mercury for more than three hours. The blood pressure was 130 systolic and 90 diastolic. How did the tissues in the dome of the uterus obtain their blood supply? The following mechanism appeared to be responsible. Between contractions adequate relaxation occurred in the body of the fundus of the uterus and in the lower uterine segment, and arterial blood could flow into vessels in these areas. During contractions the pressures in these regions exceeded that in the dome by 30 to 40 mm. of mercury (fig. 4) and squeezed some arterial blood up into the vessels of the dome since the wave of contraction passes upward toward the dome.

In monkeys during uterine contractions the areas in the regions of the juncture of the tubes and uterus have been observed to show no blanching during uterine contractions.[9] In view of the observation that uterine pressures differ at different levels within the uterus, this localized lack of blanching in the monkey could be evidence that contractions of the uterus of the monkey also serve to facilitate arterial blood flow to all portions of the uterus.

Though myometrial activity is an important factor in the distress of dysmenorrhea, the amount of edema, congestion, vasoconstriction, tissue fragility, heightened tissue irritability and fibrinolysis of menstrual débris are also important physiologic factors.

SUMMARY AND CONCLUSION

Dysmenorrhea can be produced in control patients and intensified in dysmenorrheal patients by intravenous injections of small quantities of "pitressin" and by stretching the uterus or cervix. It could not be produced with intravenous injections of acetylcholine, histamine or "pitocin" in quantities which produced definite side actions.

In some patients "pitressin" has greater uterine stimulative action than "pitocin." Hence, the present bioassay method of these preparations is misleading.

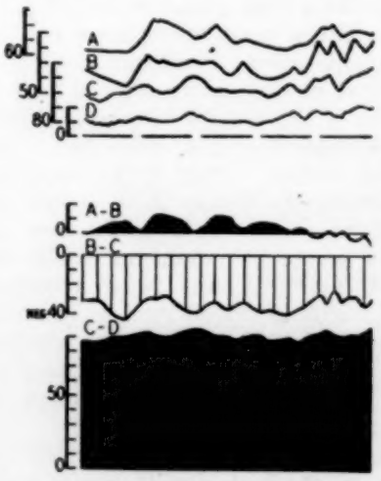

Fig. 5.—Uterine pressure curve showing constriction ring during menses: The curves were obtained from use of multiple balloons and were replotted as in figure 4. Compare curves *C* of figures 4 and 5. Note that constriction rings seem to occur during menstruation. The downward propulsion of menstrual débris is prevented by the constriction ring.

Summation, relative refractory period and incomplete and complete tetany were observed in the human uterus.

Uterine pressures at which patients complained of pain varied a great deal. When contractions were coordinated, discomfort usually occurred at a pressure near 70 to 100 mm. of mercury and distress appeared near a pressure of 140 to 180 mm. of mercury. In the presence of incomplete or complete uterine tetany, pain always was severe at much lower pressures; usually severe pain occurred with pressures of 40 to 80 mm. of mercury.

Pressure records secured from different levels of the uterus showed definite differences. The shortest con-

tractions were observed in the region of the cervix. The musculature in the dome of the fundus is slow to contract and even slower to relax. Downward toward the lower uterine segment, the musculature progressively contracts faster and relaxes faster and more completely. As a result uterine pressure levels are different at various levels within the uterus. Débris is pushed outward within the uterus during relaxation and not during contractions.

Disorganized contractions fail to propel menstrual débris downward within the uterus. In some patients the disorganized contractions develop a constriction ring which further obstructs the outflow of débris.

It was found necessary to use intrauterine electrodes to obtain electrouterograms which showed a relationship to the uterine activity.

ABSTRACT OF DISCUSSION

DR. M. PIERCE RUCKER, Richmond, Va.: No other organ in the body is so capricious as the uterus. There is a tremendous species variation in its behavior. In the human being the least change in the environment of the uterine fiber makes a difference. The behavior of the uterus varies with the time of the menstrual cycle and with the stage of pregnancy. For instance, the response of the parturient uterus to posterior pituitary injection varies greatly according to whether it is given antepartum, intrapartum or postpartum. Nor can one predict what the behavior of the intact organ will be, from the action of strips of isolated muscle. Furthermore, different parts of the uterus behave differently. This has been known to be true for the pregnant uterus for some time. So far as I know this is the first time that it has been shown to be true for the nonpregnant organ. One might pursue this line of thought further by showing how certain drugs change the response of the uterus to other drugs. I mention this to show how important are careful studies on the intact uterus, such as the report which was heard today. I was interested to see that Dr. Woodbury and his associates' work confirms that of Bickers, who showed that increased tone between contractions was found in dysmenorrhea. They have gone further and shown that the nonpregnant uterus may contract in a regular orderly manner which they called organized contractions or in a disorderly or disorganized manner. The latter type is also associated with dysmenorrhea. The orderly type, as I understood from the paper, is associated with dysmenorrhea only when the tone of the muscle is increased. They demonstrated a constriction ring in a dysmenorrheic uterus. I have been interested in constriction rings in the parturient uterus for years. In labor such rings are found in the uterus that contracts in an irregular manner: another phenomenon which the parturient and the menstruating uterus have in common. I am not surprised that they found no effect from acetylcholine. I have tried this drug in uterine inertia without noticeable benefit. Perhaps the greatest surprise, aside from the demonstration of a constriction ring in a menstruating uterus, is that the nonpregnant uterus may contract with more force than the uterus in labor: And yet when one considers the relative thickness of the wall of the pregnant and nonpregnant uterus one should not be surprised. The authors are to be congratulated on their work.

DR. A. R. ABARBANEL, Beverly Hills, Calif.: I want to join with Dr. Rucker in congratulating Dr. Woodbury and his associates for an ingenious and remarkable piece of work. They have shown, along with Moir, that a uterine contraction leading to anoxemia of the uterine muscle can be associated with severe pain. I think that they might also have gone a little further and tried to duplicate the experiments of Robertson, who showed that by injecting sterile water in a tense nervous patient, spasmodic contractions of the uterus might result. Prolonged labor may be caused by what is considered to be myometrial dyskinesia or disorganized myometrial contractions. Clinically, it has been shown by several investigators that estrogen plays a distinct and favorable role in this condition. As a matter of fact, the effect of estrogen on the uterine muscle in disorganized contractions might be compared with that of digitalis on the heart in a similar condition. I would like to raise the question of the influence of various hormonal substances. For example, it has been noticed in hypertonicity of the uterotubal junction in the human subject that methyltestosterone sublingually in the form of "linguets" would abolish the spasm of the uterotubal junction as well as reduce the hypertonicity to normal values. In cases in which spasmodic dysmenorrhea was present, dysmenorrhea was usually relieved. It is believed that this hormonal substance acts directly on the myometrium. I too have found that the pattern of uterine contractions in the pregnant uterus during labor is essentially the same as that reported by Dr. Woodbury this morning. Further, I also found that the top portion of the corpus uteri contracts first and relaxes last, while the lower portions of the corpus contract last and relax first. As a result, it is clearly evident that the contractions of the human uterus in labor are patterned so that eventual expulsion of the fetus finally results.

DR. WILLIAM E. HUNTER, Los Angeles: There are three factors that should be considered in the production of pain in dysmenorrhea: (1) the pain threshold; (2) the physiologic changes associated with ovarian activity, and (3) the sensory conditioning process. The pain threshold varies in different persons. As a rule women with dysmenorrhea have lower pain thresholds. They are more sensitive to painful stimuli during adolescence, reproductive life and the menopause. Ovulation produces certain physiologic changes, such as elevation of temperature, tissue edema, fragility of the blood vessels, irritability of the nervous system and muscular tension. All ovulating women feel these changes if they look for them. About 50 per cent are conscious of them, but only 5 per cent consult their physicians for relief. One only sees those patients who are somewhat incapacitated. The sensory conditioning process is a method of learning. By repetition one learns to read, and just so the dysmenorrheic woman learns to pick up subnormal stimuli and becomes sensitized to them. If an electrode is placed on the arm and the subject is instructed to break the circuit when pain is felt, after five hundred trials she will begin to feel pain when no stimulus is sent through the apparatus. If the experiment is repeated with stimuli above the pain threshold level, there is a conditioning to pain and in time the patient will complain of severe pain at the threshold level or even below. Is it not true that the perception and reaction to pain can be changed psychically by raising the pain threshold? I believe that many of the cures obtained by diverse types of treatments for dysmenorrhea are accomplished by suggestion. If the treatment is dramatic and the patient is led to believe in its efficacy either by injections or by dilation of the ureter, urethra or cervix, as I have done in many cases, the patient is relieved. She is temporarily cured by suggestion. For that reason I think that much of the work being done to relieve pain follows the old dictum, "Here is your trouble, now I remove it, now you are well."

Diphtheria in Partially Immune Persons.—It is probably a safe statement that the average American physician, who constituted the bulk of the medical corps since the days of mobilization, was initially not well acquainted with clinical diphtheria. His concept of the pharyngeal lesion was that of childhood diphtheria commonly found in textbooks. Diphtheria in partially immune persons, and accordingly that of many adults, is often somewhat different. During the course of the war this was probably first observed during a diphtheria epidemic in a prisoner of war camp at Aliceville, Ala. Here it was noted that "less than one third of the diseased throats were suggestive of diphtheria, while more than one half the lesions were those of follicular tonsillitis."—Schulze, Hartwin A.: Diphtheria in the U. S. Army in Europe, *M. Bull.*, June 1947.

Special Article

TYPHOID IN THE LARGE CITIES OF THE UNITED STATES IN 1946

Thirty-Third Report

In conformity with the practice adopted in previous reports a communication was addressed to the health officer of each of the cities requesting the number of deaths from typhoid, excluding paratyphoid, both among residents and among nonresidents as recorded for 1946. The health directors of many cities keep no data on reallocations for residence, and this information can be secured only from the state health department, whose figures are not available until several months after the close of the calendar year. In some instances multiple inquiries brought forth irreconcilable replies. Too frequently the reporting of deaths is left to subordinate employees, and carelessness is evident. The health officer should give final approval to statistical data released by his department. A few health officers seem to have no knowledge of how many typhoid deaths have occurred in their community, and it becomes an increasing struggle to obtain accurate data. The possession of basic data seems paramount to the establishment and operation of an adequate local health service.

For most of the cities of 100,000 population or more the Bureau of the Census has no estimates of population for dates since 1940. There are figures for a few of these cities, based on special censuses or sample censuses for various dates since 1940. Special censuses were conducted for Long Beach, Los Angeles, Oakland and San Diego in 1946. The Bureau of the Census has released a population estimate for Washington, D. C., as of July 1, 1946. Sample censuses were taken in 1944 for Detroit, Norfolk, Portland, San Francisco, Seattle and Tacoma. (A special census was taken in San Francisco in August 1945.) Estimates of the civilian population of counties are available for March 1 and Nov. 1, 1943, based on registrations for war ration books 2 and 4. Some of the cities (Baltimore, Denver, New Orleans, New York, Norfolk, Philadelphia, Richmond, St. Louis, San Francisco, Washington) are coterminous with their counties and the figures given in the releases are city as well as county estimates. Since for the vast majority of cities no official figures are available for any date subsequent to that of the 1940 federal census, it has again been deemed advisable to employ the populations determined by this uniform tabulation. For those cities for which special census figures are available, corrections in rates have been shown in footnotes. In most instances there are no or only minor changes in the rates. In no instance has any rate been extended beyond the first decimal point, and it is realized that this practice creates a slight disadvantage for the extremely large cities which now have but a single death from typhoid (Detroit).

Paratyphoid has again been excluded. Special note has been made of cities in which all deaths occur among nonresidents. In addition to the sixty-two cities enumerated on the honor roll in table 10, five cities (Birmingham, Norfolk, Pittsburgh, St. Louis, Spokane) are qualified for such acclaim but have been charged with typhoid deaths among nonresidents cared for in these cities. In five other cities (Houston, Knoxville, Los Angeles, New Orleans, Tacoma) more than one

TABLE 1.—*Death Rates of Fourteen Cities in New England States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Cambridge	0.0	0.0	0.0	0.2*	0.8	2.1	4.3	2.5	4.0	9.8
Fall River	0.0	0.0	0.0	0.2	0.2	2.2	2.3	8.5	13.4	13.5
Lynn	0.0	0.0	0.0	0.2	0.2	1.5	1.6	3.9	7.2	14.1
Springfield	0.0	0.0	0.0	0.2*	1.1†	0.4	2.0	4.4	17.6	19.9
Hartford	0.0	0.0	0.0	0.5	1.0†	1.3	2.5	6.0	15.0	19.0
Somerville	0.0	0.0	0.0	0.6	0.4	1.3	1.6	2.8	7.9	12.1
Worcester	0.0	0.0	0.1	0.2*	0.5*	1.0	2.3	3.5	5.0	11.8
Bridgeport	0.0	0.0	0.1	0.4	0.3	0.5	2.2	4.8	5.0	10.3
Lowell	0.0	0.0	0.2	0.4	1.2	2.6	2.4	5.2	10.2	13.9
Providence	0.0	0.0	0.2†	0.6	1.1†	1.3	1.8	3.8	8.7	21.5
New Haven	0.0	0.0	0.2†	1.0	0.7†	0.6	4.4	6.8	18.2	30.8
Waterbury	0.0	0.0	0.8	0.2	0.4	1.2	1.0	8.0	18.8	....
Boston	0.0	0.1	0.1	0.3†	0.6	1.2	2.2	2.5	9.0	16.0
New Bedford	0.0	0.0	0.2	0.4	1.1	1.5	1.7	6.0	15.0	16.1

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.

TABLE 2.—*Death Rates of Eighteen Cities in Middle Atlantic States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Yonkers	0.0	0.0	0.0	0.3	0.7	0.5	1.7	4.8	5.0	10.3
Erie	0.0	0.0	0.0	0.7†	1.2	0.9	2.3	6.9	49.0	46.6
Albany	0.0	0.0	0.0	1.2†	1.2	1.8	5.6	8.0	18.6	17.4
Trenton	0.0	0.0	0.0	1.4†	1.1	2.1	8.2	8.6	22.3	28.1
Jersey City	0.0	0.0	0.1†	0.6	0.3	0.9	2.7	4.5	7.2	12.6
Paterson	0.0	0.0	0.1*	0.7†	0.9	1.0	3.3	4.1	9.1	19.3
Buffalo	0.0	0.0	0.2	0.1	0.6	2.7	3.9	8.1	15.4	22.8
Reading	0.0	0.0	0.2	0.5	0.4	1.6	6.0	10.0	31.9	42.0
Syracuse	0.0	0.0	0.3†	0.3*	0.8	0.8	2.3	7.7	12.3	15.6
Elizabeth	0.0	0.0	0.4	0.5	0.9	1.6	2.4	3.3	8.0	16.6
Newark	0.0	0.5	0.1	0.3	0.4	0.9	2.3	3.3	6.8	14.6
Camden	0.0	1.7†	0.5†	1.4†	2.7	4.4	5.9	4.9	4.5	4.0
New York	0.1	0.1	0.1	0.3	0.8	1.3	2.6	3.2	8.0	13.5
Pittsburgh	0.1*	0.4	0.3	0.7	0.9	2.4	3.9	7.7	15.9	65.0
Philadelphia	0.1	0.6	0.3	0.8	0.9	1.1	2.2	4.9	11.2	41.7
Rochester	0.3	0.0	0.1	0.3†	0.4	1.7	2.1	2.9	9.6	12.8
Scranton	0.7	0.0	0.0	0.3	1.4	1.8	2.4	3.8	9.3	31.5
Utica	1.9	0.0	0.4†	0.2*	0.2	1.1	3.9‡	...	...	....

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
‡ Incomplete data.

TABLE 3.—*Death Rates of Ten Cities in South Atlantic States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Charlotte	0.0	0.0	0.0†	1.6†	2.5	...	....	....	....	....
Miami	0.0	0.6	0.6	2.4	2.0	3.5	....	....	....	....
Jacksonville	0.0	0.6	0.8	2.3	1.6	4.4	....	....	....	....
Atlanta	0.0	1.0†	0.5†	2.1†	7.3	11.1	14.5	14.2	31.4	58.4
Richmond	0.0	2.1†‖	1.3†‖	2.5†	2.4	1.9	5.7	9.7	15.7	34.0
Baltimore	0.1	0.2†	0.3†	0.9†	1.4	3.2	4.0	11.8	23.7	35.1
Washington	0.9‡	0.3¶	0.4†¶	1.1	2.2	2.8	5.4	9.5	17.2	36.7
Wilmington	0.9	0.9*	0.2*	0.5	1.5	3.1	4.7	25.8#	23.2#	33.0
Norfolk	1.4*	0.7¶	0.3¶	1.1†	3.3	2.2	2.8	8.8	21.7	42.1
Tampa	1.8	1.8	1.8	0.6†	3.4	3.8	19.1	43.5#	....	....

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
‖ 1943 special census gives rate of 1.8 for 1945 and 1.2 for 1941-1945 for Richmond.
¶ 1944 special census gives rate of 0.2 for 1945 and 0.3 for 1941-1945 for Washington and rate of 0.4 for 1945 and 0.2 for 1941-1945 for Norfolk.
‡ 1946 special census gives rate of 0.7 for 1946 for Washington.

The preceding articles in this series were published in THE JOURNAL May 31, 1913, p. 1702; May 9, 1914, p. 1473; May 17, 1915, p. 1322; April 22, 1916, p. 1305; March 17, 1917, p. 845; March 16, 1918, p. 777; April 5, 1919, p. 997; March 6, 1920, p. 672; March 26, 1921, p. 860; March 25, 1922, p. 890; March 10, 1923, p. 691; Feb. 2, 1924, p. 389; March 14, 1925, p. 813; March 27, 1926, p. 948; April 9, 1927, p. 1148; May 19, 1928, p. 1624; May 18, 1929, p. 1674; May 17, 1930, p. 1574; May 9, 1931, p. 1576; April 30, 1932, p. 1550; May 13, 1933, p. 1491; May 19, 1934, p. 1677; June 8, 1935, p. 2093; June 6, 1936, p. 1983; June 19, 1937, p. 2118; July 30, 1938, p. 414; May 13, 1939, p. 1941; May 25, 1940, p. 2103; Jan. 17, 1942, p. 222; Aug. 8, 1942, p. 1188; Aug. 21, 1943, p. 1181, and July 6, 1946, p. 817.

third of the reported deaths were stated to have been among nonresidents. It is noteworthy that for the first time there is no city with a death rate in excess of 2.0, and thus the list of cities of second rank is completely omitted.

The number of cities with no typhoid death during the past two or more years has increased from twenty-five in 1941 and forty-one in 1945 to a new high of forty-nine in 1946 (table 9). Fort Wayne continues to head the list with no death in twelve years. Fall River and Lynn report no death in ten years, Cambridge no death in nine years. Hartford, Springfield and Yonkers have clear records for eight years. South Bend reports no typhoid death among residents for eleven years.

The New England cities (table 1) (population 2,579,152) have set a new all-time record with no deaths from typhoid in 1946. These cities have reported the lowest group rates in each of the quinquennial periods (table 13). The East North Central cities (0.06), although reporting a new low rate, must now be content with second place. They were in first place

TABLE 4.—*Death Rates of Nineteen Cities in East North Central States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Fort Wayne	0.0	0.0	0.0	0.0	2.1	4.2	12.9	7.3	....	....
Canton	0.0	0.0	0.0	0.4†	0.8	1.4	3.3	8.9	....	....
Grand Rapids	0.0	0.0	0.0	0.5†	0.2	1.0	1.9	9.1	25.5	29.7
Gary	0.0	0.0	0.0	1.1	0.8	...	...	...	....	....
Evansville	0.0	0.0	0.0	1.2†	1.8	6.2	5.0	17.5	32.0	35.0
Milwaukee	0.0	0.0	0.1	0.1	0.2	0.8	1.6	6.5	13.6	27.0
Akron	0.0	0.0	0.1*	0.6†	0.9	1.5	2.4	10.6	21.0	27.7#
South Bend	0.0	0.0	0.2*	0.0	0.8	...	...	....	....	....
Cleveland	0.0	0.0	0.2	0.6	1.1	1.0	2.0	4.0	10.0	15.7
Flint	0.0	0.0	0.2	1.3†	0.8	1.6	4.6	22.7	18.8	46.9
Peoria	0.0	0.0	0.6†	1.3†	0.9	0.2	3.7	5.7	16.4	15.7#
Toledo	0.0	0.0	1.1	1.0†	1.2	3.0	5.8	10.6	31.4	37.5
Indianapolis	0.0	0.3*	0.3†	1.2†	1.8	2.7	4.6	10.3	20.5	30.4
Columbus	0.0	0.3*	0.4†	1.4†	1.9	2.1	3.5	7.1	15.8	40.0
Chicago	0.1	0.1	0.1	0.3	0.4	0.6	1.4	2.4	8.2	15.8
Detroit	0.1	0.1*	0.1	0.4	0.7	1.3	4.1	8.1	15.4	22.8
Cincinnati	0.2	0.4†	0.3†	1.1†	1.4	2.5	3.2	3.4	7.8	30.1
Dayton	0.5	0.5	0.3†	1.4	0.8	1.9	3.3	9.3	14.8	22.5
Youngstown	0.6	0.0	0.1*	0.7	1.2	1.1	7.2	19.2	29.5	35.1

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.

in 1945 (0.07). The West North Central cities follow close behind (0.07) and nose out the Middle Atlantic cities (0.11). Only the Mountain and Pacific cities record a higher death rate for 1946 (0.31) than for 1945 (0.17). There have been but three deaths in the New England cities during the past three years, one in Providence in 1944, one each in Boston and New Bedford in 1945. While in 1940 one half of the eighteen cities which recorded no deaths from typhoid during a two year period were to be found among the New England cities, in 1946 only about one fourth (fourteen among forty-nine) were from this group. The New England cities with a perfect record can do no better, but there is a tendency for other cities to attain the high standards set by this group.

Twelve of the large Middle Atlantic cities (population 13,129,185) (there were thirteen in 1945) report no death from typhoid in 1946 (table 2). Albany, Buffalo, Elizabeth, Erie, Jersey City, Paterson, Reading, Syracuse, Trenton and Yonkers have maintained their ranking of 1945 while Camden and Newark have been added to the honor roll. Rochester and Scranton, each with one death in 1946, were eliminated from the honor roll, Scranton after six years with no death. The Middle Atlantic cities have a group rate (0.11) which is but slightly higher than that of the East North Central and West North Central cities. It is much lower than the rate for 1945 (0.23) but not as low as that for 1943 (0.08). In 1946 there were fourteen deaths, thirteen among residents; in 1945 there were thirty,

TABLE 5.—*Death Rates of Six Cities in East South Central States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Chattanooga	0.0	0.0	0.2	0.9	5.8	8.0	18.6	27.2	35.8#	....
Nashville	0.0	0.6*	0.5*	3.4†	5.7	18.2	17.8	20.7	40.2	61.2
Memphis	0.3	0.0	0.3†	4.0†	7.5	9.3	18.9	27.7	42.5	35.3
Louisville	0.6	0.6†	0.5†	0.9	2.8	3.7	4.9	9.7	19.7	52.7
Birmingham	0.7*	1.9†	0.9†	2.5†	4.1	8.0	10.8	31.5	41.3	41.7
Knoxville	1.8†	0.9*	1.2*	3.8	6.0	10.7	20.8	25.3#	....	....

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.

twenty-five among residents; in 1943 there were eleven, nine among residents. Jersey City reports no typhoid death for four years (table 9), Paterson and Reading none for five years, Albany and Trenton none for six years, Erie none for seven years, Yonkers none for eight years. Utica records one death among residents in 1946, the second such death in fifteen years. It is stated that although the death occurred in a resident of Utica, the source of infection was reliably established as being in Italy. New York records eight deaths in 1946, all among residents (there were eleven in 1945, thirteen in 1944). The low mark was reached in New York in 1943 with four deaths, three among residents. (In 1942 there were five, all among residents.) New York reports a localized outbreak with 20 cases and no deaths in August 1946; the outbreak was attributed to fresh vegetables contaminated by sewage backed up out of a toilet bowl in an apartment immediately over the vegetable store. It is stated that in Philadelphia there occurred two deaths, both among residents. This marks a new low for this city. Pittsburgh records but

TABLE 6.—*Death Rates of Nine Cities in West North Central States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Duluth	0.0	0.0	0.0	0.2	1.0	1.1	1.7	4.4	19.8	45.5
Des Moines	0.0	0.0	0.0	1.3	2.5	2.4	2.2	6.4	15.9	23.7
Minneapolis	0.0	0.0	0.1	0.2	0.8	0.8	1.9	5.0	10.6	32.1
Kansas City, Mo.	0.0	0.0	0.1†	0.9†	1.6	2.8	5.7	10.6	16.2	35.6
Wichita	0.0	0.0	0.2	0.4	1.1	1.2	6.3	....	....	....
Kansas City, Kan.	0.0	0.0	0.8†	1.0	1.0	1.7	5.0	9.4	31.1	74.5#
St. Paul	0.0	0.3	0.1	0.3	0.7	1.4	3.4	3.1	9.2	12.8
St. Louis	0.1*	0.1	0.2†	0.7	1.6	2.1	3.9	6.5	12.1	14.7
Omaha	0.4	0.4	0.1	0.6	0.9	1.3	3.3	5.7	14.9	40.7

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.

one death, which occurred among nonresidents. Pittsburgh reported no death among residents in 1942 and again in 1946.

The rate (0.44) for the South Atlantic cities (table 3) (population 2,727,985) is twice the low rate for 1944 (0.22) but is significantly lower than the rate for 1945 (0.62). In these cities there occurred twelve deaths in 1946 (nine among residents), seventeen in 1945 (eleven among residents), six in 1944 (three

among residents). Five cities (Atlanta, Charlotte, Jacksonville, Miami, Richmond) are on the honor roll, and it is stated that the two deaths in Norfolk occurred among nonresidents. In 1945 Charlotte was the only city on the honor roll. In 1944 there were six such cities (Atlanta, Jacksonville, Miami, Norfolk, Tampa, Wilmington). Charlotte has been included in the group for the fifth time; however, for purposes of adequate comparison the figures for this city have been omitted in calculating rates for the group as a whole. Washington records six deaths in 1946, five among residents (there were two deaths in 1945, four in 1944, one in 1943). Tampa reports two deaths both among residents, Baltimore and Wilmington each one such death.

TABLE 7.—*Death Rates of Eight Cities in West South Central States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Tulsa	0.0	0.0	0.0	0.4	1.1	8.3	16.2#	....	....	....
Oklahoma City	0.0	0.0	0.1	2.6	4.3	7.4#	....	....	....	....
El Paso	0.0	0.0	2.7	4.5	5.2	9.1	10.8	30.7	42.8	....
San Antonio	0.4	0.4	0.3	2.9	4.3	4.6	9.3	23.3	29.5	35.9
New Orleans	0.4†	1.2*‖	1.4†‖	5.0†	9.6	9.9	11.6	17.5	20.9	35.6
Houston	0.5†	0.8†	1.1	2.3	3.2	4.8	7.6	14.2	38.1	49.5#
Fort Worth	0.6	0.6	0.4†	2.1	4.5	5.9	6.1	16.3#	11.9	27.8
Dallas	0.7	0.3	0.9†	2.7†	5.1	7.3	11.2	17.2	....	....

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
‖ 1943 special census gives rate of 1.1 for 1945 and 1.3 for 1941-1945 for New Orleans.

The East North Central cities (population 9,386,378) have lost first place, which rank they held in 1945 and back in 1941 and 1938. Fourteen (thirteen exclusive of Gary) of the cities in this group (Akron, Canton, Cleveland, Columbus, Evansville, Flint, Fort Wayne, Gary, Grand Rapids, Indianapolis, Milwaukee, Peoria, South Bend, Toledo) report no death from typhoid in 1946 (table 4). Gary was added to this group in 1940, but figures for this city have been omitted in determining rates for the group as a whole. The number of typhoid deaths decreased from seven in 1945 (thirty-three in 1940, thirteen in 1944) to six in 1946, a new low (the rate from 0.35 in 1940, 0.14 in 1944 and 0.07 in 1945 to 0.06 in 1946). Twelve cities (Akron, Canton, Cleveland, Evansville, Flint, Fort Wayne, Gary, Grand Rapids, Milwaukee, Peoria, South Bend, Toledo) record no typhoid death during 1945 and 1946 (table 9), Fort Wayne none for twelve years (the longest honor record of any city), Canton and Grand Rapids none for seven years, Evansville and Gary none for six years. Chicago records two deaths and Cincinnati, Dayton, Detroit and Youngstown each one death, all among residents. Detroit reports that among 24 cases of typhoid attributed to a church supper in a nearby community, 13 were in residents of Detroit among whom there occurred no deaths. A person preparing and serving food was found to be a typhoid carrier.

TABLE 8.—*Death Rates of Twelve Cities in Mountain and Pacific States from Typhoid per Hundred Thousand of Population*

	1946	1945	1941-1945	1936-1940	1931-1935	1926-1930	1921-1925	1916-1920	1911-1915	1906-1910
Salt Lake City	0.0	0.0	0.1*	0.3*	0.7	1.9	6.0	9.3	13.2	41.1
Long Beach	0.0	0.0	0.1	0.5	0.5	1.1	2.1#	...	....	....
Oakland	0.0	0.0	0.2	0.5†	1.2	1.2	2.0	3.8	8.7	21.5
San Diego	0.0	0.0	0.2†‖	0.8	1.3	1.0	1.6	7.9	17.0	10.8
Seattle	0.0	0.3	0.1	0.2	0.7	2.2	2.6	2.9	5.7	25.2
Denver	0.3	0.0	0.3†	1.4	1.7	2.6	5.1	5.8	12.0	37.5
San Francisco	0.3	0.2*¶	0.1†	0.4	0.8	2.0	2.8	4.6	13.6	26.3
Los Angeles	0.3†	0.3	0.3	0.7†	0.8	1.5	3.0	3.6	10.7	19.0
Portland	0.7	0.0	0.1*	0.3	0.8	2.3	3.5	4.5	10.8	23.2
Spokane	0.8*	0.0	0.6†	1.2	1.0	2.2	4.4	4.9	17.1	50.3
Sacramento	0.9	0.9	0.4†	1.5*	0.3	...	...	...	....	....
Tacoma	1.8†	0.0	0.5†	0.5	0.9	1.8	3.7	2.9	10.4	19.0

* All typhoid deaths were stated to be in nonresidents.
† One third or more of the reported typhoid deaths were stated to be in nonresidents.
# Incomplete data.
‖ 1944 special census gives rate of 0.1 for 1941-1945 for San Diego.
¶ 1945 special census gives rate of 0.1 for 1945 for San Francisco.

The six cities (table 5) in the East South Central group (population 1,286,747) show a decrease in the death rate (0.54 in 1946, 0.70 in 1945). However, a lower death rate (0.31) was recorded in 1944 and the same rate in 1942 (0.54). This group has the highest group rate for 1946, a position which it also held in 1945, the South Atlantic cities being next (0.44). The effect of hospitalizing patients from surrounding rural areas continues to be evident. Of seven deaths in 1946, four were among nonresidents (seven of nine deaths in 1945). Two cities (Chattanooga and Nashville) report no deaths among either residents or nonresidents and appear on the honor roll. The two deaths in Birmingham and the one death in Memphis were stated to be among nonresidents. Chattanooga reports no death for five years (table 9). Knoxville records two deaths, one among residents; Louisville two deaths, both among residents.

TABLE 9.—*Forty-Nine Cities with No Typhoid Deaths in 1945 and 1946 #*

Akron 12	Fort Wayne 12	Providence 10
Albany 6	Gary 6	Reading 5
Bridgeport 5	Grand Rapids 7	Salt Lake City 5
Buffalo 10	Hartford 8	San Diego 4
Canton 7	Jersey City 4	Somerville 6
Cambridge 9	Kansas City, Kan. 3	South Bend 12
Charlotte 9	Kansas City, Mo. 10	Springfield 8
Chattanooga 5	Long Beach 3	Syracuse 10
Cleveland 12	Lowell 3	Toledo 3
Des Moines 7	Lynn 10	Trenton 6
Duluth 8	Milwaukee 3	Tulsa 7
Elizabeth 10	Minneapolis 5	Waterbury 3
El Paso 9	New Haven 3	Wichita 4
Erie 7	Oakland 3	Worcester 5
Evansville 6	Oklahoma City 10	Yonkers 8
Fall River 10	Paterson 5	
Flint 12	Peoria 3	

12 No typhoid deaths in twelve years.
10 No typhoid deaths in ten years.
9 No typhoid deaths in nine years.
8 No typhoid deaths in eight years.
7 No typhoid deaths in seven years.
6 No typhoid deaths in six years.
5 No typhoid deaths in five years.
4 No typhoid deaths in four years.
3 No typhoid deaths in three years.
Forty-seven without Charlotte and Gary.

The West North Central cities (population 2,716,484) show a decrease in the number of deaths from three to two (table 6); one of the two was among residents. The death rate declined from 0.11 in 1945 (it was the same in 1943 and 1944) to 0.07 in 1946. Seven cities (Des Moines, Duluth, Kansas City, Kan., Kansas City, Mo., Minneapolis, St. Paul, Wichita) record no death, and St. Louis reports one death among nonresidents. After a period of four years (1941 to 1944) without a death, Omaha reported one death in 1945 and another in 1946. There has occurred no typhoid death in Duluth for the past eight years (table 9), none in

Des Moines for seven years, none in Minneapolis for five years, none in Wichita for four years. Kansas City, Mo., records no death among residents for five years.

The eight cities of the West South Central group (table 7) (population 2,048,692) report a continued reduction in the death rate (2.00 in 1940, 0.83 in 1943, 0.59 in 1945, 0.39 in 1946). The actual number of deaths decreased from twelve in 1945 (forty-one in 1940) to eight in 1946, the lowest of record. Of the eight deaths in this group of cities, two were among nonresidents (eight of twelve in 1945). The low point for deaths among residents (four) occurred in 1945. Three cities (El Paso, Oklahoma City, Tulsa) record no death in 1946. Tulsa reports no death for seven years. Dallas reports two deaths among residents. It is stated that of the two deaths each in Houston and New Orleans, one each was among nonresidents. Fort Worth and San Antonio record one death each among residents. Noteworthy is the reduction in the death rate of the cities of this group from 5.36 for the quinquennial period 1931 to 1935 to a new low of 0.39 in 1946.

The eleven cities (excluding Sacramento) in the Mountain and Pacific states (table 8) (population 4,186,039) report an increase from seven in 1945 to thirteen in 1946 (there were but five deaths in 1944). The rate has increased from 0.17 to 0.31. (It was 0.12 in 1944.) This is the only group of cities which records more deaths from typhoid in 1946 than in 1945. Sacramento has again been omitted in calculating the total number of deaths and the rates. This city reports one death among residents. Five of the Mountain

TABLE 10.—*Death Rates from Typhoid in 1946*

Honor Roll: No Typhoid Deaths (Sixty-Two Cities)‡

Akron	Fort Wayne	Paterson
Albany	Gary	Peoria
Atlanta	Grand Rapids	Providence
Boston	Hartford	Reading
Bridgeport	Indianapolis	Richmond
Buffalo	Jacksonville	Salt Lake City
Canton	Jersey City	San Diego
Cambridge	Kansas City, Kan.	Seattle
Camden	Kansas City, Mo.	Somerville
Charlotte	Long Beach	South Bend
Chattanooga	Lowell	Springfield
Cleveland	Lynn	St. Paul
Columbus	Miami	Syracuse
Des Moines	Milwaukee	Toledo
Duluth	Minneapolis	Trenton
Elizabeth	Nashville	Tulsa
El Paso	Newark	Waterbury
Erie	New Bedford	Wichita
Evansville	New Haven	Worcester
Fall River	Oakland	Yonkers
Flint	Oklahoma City	

First Rank: From 0.1 to 1.9 Deaths per Hundred Thousand (Thirty-Four Cities)¶

Baltimore	0.1	Rochester	0.3	Dallas	0.7
Chicago	0.1	San Francisco	0.3	Portland	0.7
Detroit	0.1	New Orleans	0.4†	Scranton	0.7
New York	0.1	Omaha	0.4	Spokane	0.8*
Philadelphia	0.1	San Antonio	0.4	Sacramento	0.9
Pittsburgh	0.1*	Dayton	0.5	Washington	0.9‡
St. Louis	0.1*	Houston	0.5†	Wilmington	0.9
Cincinnati	0.2	Fort Worth	0.6	Utica	1.0
Denver	0.3	Louisville	0.6	Norfolk	1.4*
Los Angeles	0.3†	Youngstown	0.6	Knoxville	1.8†
Memphis	0.3	Birmingham	0.7*	Tacoma	1.8†
				Tampa	1.8

* All typhoid deaths are stated to be in nonresidents.

† One third or more of the reported typhoid deaths were stated to be in nonresidents.

‡ Sixty without Charlotte and Gary.

¶ Thirty-three without Sacramento.

‡ 1946 special census gives rate of 0.7 for 1946 for Washington.

TABLE 11.—*Number of Cities with Various Typhoid Death Rates*

	No. of Cities	10.0 and Over	5.0 to 9.9	2.0 to 4.9	1.0 to 1.9	0.1 to 0.9	0.0
1906-1910	77	75	2	0	0	0	0
1911-1915	79	58	19	2	0	0	0
1916-1920	84	22	32	30	0	0	0
1921-1925	89	12	17	48	12	0	0
1926-1930	92	3	10	30	37	12	0
1931-1935	93	0	6	17	28	42	0
1930	93	2	6	30	23	22	10
1931	93	2	6	23	28	22	12
1932	93	1	7	13	29	29	14
1933	93	0	7	18	19	33	16
1934	93	0	9	11	27	23	23
1935	93	0	7	15	18	29	24
1936	93	0	3	15	21	36	18
1937	93	0	1	13	26	26	27
1938	93	0	3	13	14	34	29
1939	93	0	3	7	17	32	34
1940*	93	0	0	12	12	30	39
1941*	93	0	0	4	7	46	36
1942*	93	0	1	3	5	34	50
1945*	93	0	0	1	5	31	56
1946*	93	0	0	0	5	28	60

* Charlotte, Gary and Sacramento omitted.

TABLE 12.—*Total Typhoid Rate for Seventy-Eight Cities, 1910-1946**

	Population	Typhoid Deaths	Typhoid Death Rate per 100,000
1910	22,573,435	4,637	20.54
1911	23,211,341	3,950	17.02
1912	23,835,399	3,132	13.14
1913	24,457,989	3,285	13.43
1914	25,091,112	2,781	11.08
1915	25,713,346	2,434	9.47
1916	26,257,550	2,191	8.34
1917	26,865,408	2,016	7.50
1918	27,086,690	1,824†	6.73
1919	27,735,083†	1,151†	4.15
1920	28,244,878	1,088	3.85
1921	28,859,062	1,141	3.95
1922	29,473,246	963	3.26
1923	30,087,430	950	3.16
1924	30,701,614	943	3.07
1925	31,315,598	1,079	3.44
1926	31,929,782	907	2.84
1927	32,543,966	648	1.99
1928	33,158,150	628	1.89
1929	33,772,334	537	1.59
1930	34,410,235	554	1.61
1931	34,508,750	563	1.63
1932	34,607,505	442	1.28
1933	34,708,945	423	1.22
1934	34,833,650	413	1.19
1935	35,005,351	348	0.99
1936	35,196,325	337	0.96
1937	35,386,380	289	0.82
1938	35,578,011	257	0.72
1939	35,767,022	232	0.65
1940	35,895,638	172	0.48
1941	35,895,638††	123	0.34
1942	35,895,638††	83	0.23
1943	35,895,638††	78	0.22
1944	35,895,638††	66	0.18
1945	35,895,638††	80	0.22
1946	35,895,638††	54	0.15
	Rates for Ninety-Three Cities		
1935	37,025,179	385	1.04
1936	37,241,414	366	0.98
1937	37,459,339	324	0.86
1938	37,680,155	298	0.79
1939	37,900,354	259	0.68
1940	38,060,662	190	0.50
1941	38,060,662††	141	0.37
1942	38,060,662††	95	0.25
1943	38,060,662††	85	0.22
1944	38,060,662††	73	0.19
1945	38,060,662††	87	0.23
1946	38,060,662††	62	0.16

* The following fifteen cities are omitted from this table because data for the full period are not available: Canton, Chattanooga, Dallas, Fort Wayne, Jacksonville, Knoxville, Long Beach, Miami, Oklahoma City, South Bend, Tampa, Tulsa, Utica, Wichita, Wilmington.

† Data for Fort Worth lacking.

†† 1940 census figures used.

and Pacific cities (Long Beach, Oakland, Salt Lake City, San Diego, Seattle) report no death from typhoid in 1946. There were nine cities on the honor roll in 1944, eight in 1945. Long Beach, Oakland, Salt Lake City and San Diego have maintained their ranking of 1945. Salt Lake City reports no death during the past five years, San Diego none for four years. It is stated that the one death in Spokane was among nonresidents. Of five deaths in Los Angeles, four were among residents. San Francisco and Tacoma each report two deaths, one among nonresidents. Denver records one death among residents.

THE HONOR ROLL

The number of cities with no death from typhoid has increased from fifty-six to sixty (table 11). Charlotte and Gary, not included in the group of ninety-three cities, also report no death. This is by far the best showing, and there remains no city with a rate of 2.0 or more. The number of cities with rates of less than 1.0 has increased by one (eighty-eight in 1946). Forty-seven cities (forty-nine with Charlotte and Gary) record no typhoid death in 1945 and 1946 (table 10). It is again emphasized that five cities (Birmingham, Norfolk, Pittsburgh, St. Louis, Spokane) in the first rank would appear in the honor roll were they not charged with deaths among nonresidents.

TABLE 13.—*Total Typhoid Death Rate per Hundred Thousand of Population for Ninety-Three Cities According to Geographic Divisions*

	Population*	Typhoid Deaths 1946	Typhoid Deaths 1945	Typhoid Death Rates 1946	1945	1941-1945	1936-1940	1931-1935
New England	2,579,152	0	2	0.00	0.08	0.14	0.39	0.70
Middle Atlantic	13,129,185	14	30	0.11	0.23	0.17	0.43	0.80
South Atlantic	2,727,985	12	17	0.44	0.62	0.54	1.14	2.70
East North Central	9,386,378	6	7	0.06	0.07	0.16	0.53	0.75
East South Central	1,286,747	7	9	0.54	0.70	0.57	2.54	4.81
West North Central	2,716,484	2	3	0.07	0.11	0.16	0.60	1.24
West South Central	2,048,692	8	12	0.39	0.59	0.89	3.09	5.36
Mountain and Pacific	4,186,039	13	7	0.31	0.17	0.23	0.60	0.88

* 1940 census figures used.

For the seventy-eight cities (table 12) for which data are available since 1910 there occurred fifty-four deaths from typhoid in 1946 (eighty in 1945, sixty-six in 1944 and seventy-eight in 1943). This provides a new low with a rate of 0.15 (the previous low was 0.18 in 1944). For the ninety-three cities the number of deaths in 1946 is sixty-two (eighty-seven in 1945 and seventy-three in 1944).

Table 13 shows the number of deaths for each of the past two years in each group of cities. It also shows the rates for 1945 and 1946 and the quinquennium 1941 to 1945 and the two preceding five year periods. It is hoped that the New England cities will continue to hold their unique record with no deaths and that other cities will soon find themselves in the same class. The continued reduction in rates for all groups is most impressive. For 1946 the health officers report only a few localized outbreaks of typhoid. Improvement has been general throughout the country, and typhoid as a cause of death in these cities is fast approaching the vanishing point. The war years do not appear to have contributed materially to the typhoid problem in the large cities.

Clinical Notes, Suggestions and New Instruments

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS

Report of Complete Recovery in Case with Multiple Pulmonary Abscesses Complicating Pregnancy

EDWARD A. BRETHAUER Jr., M.D.
Pittsburgh

From a review of the literature it is evident that thrombophlebitis of the cavernous sinus is not a rarity. It has been reported in the literature since 1818,[1] and until recently it has been 98 per cent fatal. There have been reported instances of recovery since 1869. Some of these recoveries have occurred spontaneously while others have been the result of surgical intervention, which has been a therapeutic procedure since 1902. In all patients who were cured the convalescence was greatly prolonged. Eagleton[2] reported a series of 25 cases in which there were 4 recoveries, in 3 of which the patient had surgical therapy. From the late nineteenth century until the onset of chemotherapy 58 recoveries have been reported.[3] The exact percentage of recoveries since the onset of chemotherapy is not available, but certainly reports of recovery are much more frequent in the literature.[4]

As far as the complications of thrombophlebitis of the cavernous sinus are concerned, there have been reports of meningitis, abscess of the brain, necrosis of the pituitary body and also of multiple cerebral infarcts.[5] Although it is likely that cases complicated by multiple pulmonary abscesses as a result of septic emboli have occurred, such reports are not available in American medical literature.

The etiology of thrombophlebitis of the cavernous sinus has been a subject of considerable discussion. There are usually four ways of infection:

1. Anterior foci of infection: These include the upper lip, alae nasi, vestibule of the nose and eyelids. The route of infection is by way of the angular, supraorbital and supratrochlear veins which drain into the cavernous sinus by way of the ophthalmic vein.

2. Internal foci of infection: The sources for this type of infection are the sinuses, turbinates and septum. This infection drains into the cavernous sinus by way of the sphenoid, ethmoid and ophthalmic veins.

3. Inferior foci of infection: The sources of this type are peritonsillar abscesses, operations on the tonsils, operations on the maxilla and deep cervical abscesses. These are carried by way of the pterygoid plexus or by direct proximal extension into the internal jugular through the lateral sinus and petrosal sinus.

4. Posterior foci of infection: These originate in the middle ear and mastoids. They extend from the lateral sinus through

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Written under the direction of the Research Committee of the University of Pittsburgh School of Medicine.

1. Abercrombie, J.: Observations on Chronic Inflammation of the Brain and Its Membranes, Edinburgh M. & S. J. **14:** 265-328 (July 1) 1818.

2. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses, New York, The Macmillan Company, 1926; The Carotid Plexus as the Path of Infection in Thrombosis of the Cavernous Sinus, Arch. Surg. **15:** 275-287 (Aug.) 1927.

3. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: Thrombophlebitis of the Cavernous Sinus: A Review of Reported Recoveries with Special Reference to Thrombophlebitis of Staphylococcic Origin, Arch. Ophth. **29:** 231-257 (Feb.) 1943.

4. Conte, A. J.: A Case of Cavernous Sinus Thrombophlebitis with Staphylococcic Septicemia Treated with Penicillin Resulting in Complete Recovery, Laryngoscope, **55:** 128-132 (March) 1945. MacNeal, W. J., and Cavolla, M. E.: Streptococcic Bacteremia and Afferent Thrombosis of the Cavernous Sinus with Recovery, J. A. M. A. **109:** 2139 (Dec. 25) 1937. Henner, R., and Ridall, E. G.: Thrombosis of Cavernous Sinus Treated with Penicillin and Heparin, Arch. Otolarng. **41:** 295-297 (April) 1945. Wolfe, W. C., and Gain, J. F.: Thrombosis of Cavernous Sinuses, ibid. **41:** 79-84 (Jan.) 1945. Nicholson, W. M., and Anderson, W. B.: Penicillin in the Treatment of Cavernous Sinus Thrombophlebitis, J. A. M. A. **126:** 12-15 (Sept. 2) 1944. Sears, T. P., and Wilson, F. L.: Recovery from Cavernous Sinus Thrombosis and Staphylococcic Pneumonia by Combined Use of Penicillin, Sulfadiazine and Antistaphylococcus Serum: A Case Report, Rocky Mountain M. J. **42:** 838 (Nov.) 1945.

5. Weisman, A. D.: Cavernous Sinus Thrombophlebitis: Report of Case with Multiple Cerebral Infarcts and Necrosis of Pituitary Body, New England J. Med. **231:** 119-122 (July 27) 1944.

the petrosal sinus. The most commonly observed cause for thrombophlebitis of the cavernous sinus is sinusitis, usually an acute exacerbation of a chronic sinusitis.[6]

There may be two forms of this disease. The acute form occurs as the result of a sudden occlusion. The second occurs as the result of a gradual occlusion, which allows for the development of collateral circulation. This type is usually not accompanied by the occlusive phenomena which accompany the former type. Both these forms are usually infectious in origin. The predominating organisms are either staphylococci or streptococci. It is possible to have a noninfective type of thrombosis of the cavernous sinus. This type occurs as the result of stasis of circulation due to some pathologic lesion which causes pressure on the cavernous sinus or trauma, such as a fracture of the basal portion of the skull.

From the point of view of pathology the lesion in thrombosis of the cavernous sinus is rather simple. As a result of inflammatory reaction the intima of the vein swells and slows the flow of blood, resulting in thrombus formation. The role of the pulsating internal carotid artery passing through the cavernous sinus must also be considered. This pulsation may aid in spreading the inflammation or infection and also may interfere with the flow of the blood, in that it may produce a to and fro movement.

The signs and symptoms occur usually as a result of occlusion or partial occlusion of the cavernous sinus and as a result of sepsis. There are usually pain in the orbit, headache, conjunctival edema, chemosis, ocular proptosis, dilatation of the retinal veins and partial to complete ophthalmoplegia. As a result of sepsis there are chills, fever, sweating, nausea and vomiting. One striking symptom which differentiates this condition from meningitis and other infections of brain or meninges is that the mentality is unimpaired until late in the disease.

The treatment consists in control of the infection and sepsis by means of chemotherapy and in an attempt to prevent thrombosis or at least to prevent the extension of a thrombosis. This is accomplished mainly by the use of anticoagulants. From recent literature it is apparent that combined treatment using the two foregoing principles is the most acceptable. However, cures have been reported in cases in which no anticoagulants have been employed. The case which I am reporting combined the use of anticoagulants, sulfonamide drugs and penicillin.

REPORT OF A CASE

Mrs. S. D., a 27 year old primipara three and one-half months pregnant, was admitted to the Elizabeth Steel Magee Hospital on Oct. 11, 1945 with chief complaints of severe headache, occipital pain, fever, moderate chills, nausea and vomiting. She was well until about two weeks before admission to the hospital, when there developed an infection of the upper part of the respiratory tract, for which she received symptomatic treatment at home. After the fifth day of the illness the headache became more severe and she began to have visual disturbances, nausea and vomiting. These symptoms continued for three days; then there developed a rather high fever with chills and increased sweating. At the same time the right eyelid began to swell and in several days the eye began to protrude and the lid became so swollen that the patient was unable to open the eye.

Physical examination at the time of admission revealed swelling and pronounced tenderness over the right frontal area, edema of the right conjunctiva with proptosis of the right eye. The examination of the fundi revealed blurring of the disk and dilatation of the retinal veins in the right eye. There were no significant observations in the left eye. There was a partial ophthalmoplegia of the right eye. There was definite evidence of sinus involvement, with purulent exudate coming from both nares. There was some swelling of the right occipital area but definite tenderness was absent. Examination of the ears revealed essentially normal conditions. The lungs were clear. The heart revealed no abnormalities. The heart rate was 110 to 120. The blood pressure was 130 systolic and 72 diastolic. Abdominal examination revealed a three months pregnant uterus. The reflexes were physiologic. The extremities showed no abnormalities. On admission the temperature was 100 F., but it soon rose to 104 F.

Heparin and penicillin were administered intravenously, and sulfadiazine was given by mouth. Attempts were made to keep the coagulation time between 12 and 15 minutes. Except for evidence of some involvement of the left eye, there was definite improvement during the first twenty-four hours of treatment. The temperature subsided to 101 F.; the leukocyte count dropped from 25,000 to 12,000 and the polymorphonuclear count from 92 to 78 per cent. Heparin therapy was continued for seven days. The administration of penicillin and sulfonamide drugs was continued for fourteen days, the patient receiving a total of 7,325,000 units of penicillin plus 1,080 grains (65.3 Gm.) of sulfadiazine. Four transfusions of 250 cc. of whole blood were given because of the rapid development of secondary anemia, which was probably due to toxicity and hemolytic staphylococci and streptococci.

After five days of intravenous penicillin therapy the patient had a generalized edema, became irrational and had an increased pulse rate and increased respiratory rate. Associated with this there was a moderate pulmonary edema and a gallop rhythm. The patient became comatose within the next twenty-four hours, after which she had a generalized convulsion. She was given two injections of 50 per cent magnesium sulfate, and all intravenous therapy was discontinued. She was given digitoxin because of the possibility of left ventricular failure. Within twenty-four hours she had a rather well defined diuresis; she became rational, and the pulmonary edema gradually subsided. There was a slow, continued improvement in the patient's general condition, and she was discharged on Nov. 5, 1945.

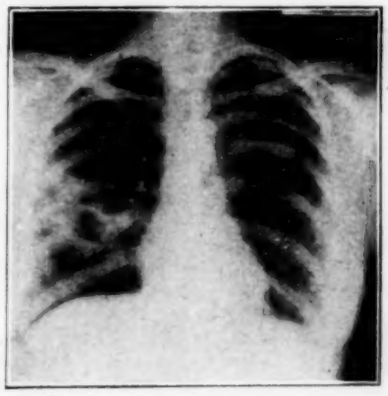

Fig. 1.—Multiple abscesses of the lungs.

While the patient was hospitalized, roentgen examination of the nasal accessory sinuses showed a suppurative sinusitis of the left maxillary, frontal and ethmoid sinuses. There was no evidence of osteomyelitis. Blood chemistry studies were within normal limits. The Kahn test was negative. Urinalyses were normal except for slight albuminuria at the time of the high fever. Blood counts showed a rapidly progressive, secondary anemia and moderate leukocytosis with a shift to the left. One blood culture before chemotherapy was negative. The culture from the sinuses showed Staphylococcus aureus and hemolytic streptococci.

About one week after discharge from the hospital, following a treatment for her sinuses, the patient had frontal headaches, moderate fever and nausea and vomiting. She was readmitted on Nov. 16, 1945, at which time she was treated for an acute exacerbation of left frontal and maxillary sinusitis. She received 700,000 units of penicillin. At the time of discharge, Nov. 26, 1945, a second roentgen examination of her sinuses revealed no suppuration. There was evidence of only hyperplastic sinusitis of the left frontal and maxillary sinuses. During this period of hospitalization, there were no residual signs of the thrombosis of the cavernous sinus except for slight blurring of both nerve heads, more decided in the right eye.

The patient was free of symptoms except for a postnasal drip, which persisted for the next four weeks. She then complained of a chronic cough, which was paroxysmal and productive of a purulent sputum. These symptoms continued until Feb. 4, 1946 when the patient came to the office for an examination. Physical observations then revealed a patient, chronically ill in appearance, with a low grade fever, tachycardia, a slight purulent nasal discharge, increased tactile fremitus and crepitant rales over the lower lobe of the right lung. At this examination there were no residual signs of the thrombosis of the cavernous sinus.

6. Jackson, C., and Jackson, C. L.: Diseases of Nose, Throat, and Ear, Philadelphia, W. B. Saunders Company, 1945, pp. 68-70.

A roentgenogram of the chest (fig. 1) showed pneumonitis with a small cavity in the lower lobe of the right lung and two cavities in the upper and lower lobes of the left lung. The cavity in the upper lobe of the left lung had a definite fluid level.

The patient was again admitted to the hospital on Feb. 25, 1946 and was given intensive penicillin therapy, for a total of 11,300,000 units. During this hospital admission she was delivered of a normal male infant that weighed 6 pounds 7 ounces (2,920 Gm.). Labor was induced by artificial rupture of the membranes, and the time of labor was twenty-one hours. The delivery was accomplished by pudendal block and a low midforceps application. Penicillin was continued post partum, and the patient had an uneventful postpartum course. At the time of discharge from the hospital, March 30, 1946, there were no abnormal physical signs on examination of the lungs, and a roentgenogram (fig. 2) showed a complete disappearance of all the cavities. There were only residual fibrous tissue changes in the right lower and left upper lobes.

COMMENT

This case is unusual and interesting in that it presents two disease states which were almost 100 per cent fatal previous to the onset of chemotherapy. Of course, the question of accuracy in diagnosis of thrombophlebitis of the cavernous sinus in such a case is always apt to be questioned. The physical observations in this patient were sufficiently definite to make the diagnosis fairly certain, but it was made even more definite by the development of embolic pulmonary abscesses. The distribution of these abscesses would certainly indicate that they were blood-borne and did not result from the drainage of infectious material from the sinuses. The absence of a positive blood culture does not rule this out. Only one blood culture was obtained, and this may have been obtained when there was no septicemia. Further blood cultures were not studied because of the extensive chemotherapy which this patient was receiving.

Fig. 2.—Complete disappearance of pulmonary abscesses.

One outstanding feature of this case was the development of massive edema after five days of intravenous penicillin and heparin therapy. By calculating the sodium intake and observing blood studies, the role of possible excessive intake of sodium in producing this massive edema was ruled out. Other factors, therefore, were responsible for this edema. The effect of sepsis and toxicity on capillary permeability and nephritic function was a possible explanation; however, at the time of the edema the toxicity and sepsis had greatly abated. One must keep in mind the likelihood of an increased amount of posterior pituitary antidiuretic factor. This could have been associated with pregnancy, or it might have been influenced by the cavernous sinus disease. Because of the latter, the secretions of the pituitary may have been increased by irritation of the secretory cells as a result of either an inflammatory reaction or an impairment of circulation. More involved blood chemistry and water balance studies might have clarified the cause of the massive edema. In my opinion all these factors played a role and that of the antidiuretic factor should be emphasized.

The amount of penicillin used in this case was certainly extremely large. It was considered at the time that the patient was treated for the pulmonary abscesses that it would be advisable to use exceedingly large doses of penicillin because she had received moderately large doses to treat the thrombosis of the cavernous sinus. It was assumed that the same organism which caused the thrombosis was responsible for the abscesses and that possibly this organism might have developed some resistance to penicillin. This reasoning was prompted by the fact that septic emboli and pulmonary abscesses developed even though the patient was getting moderately large doses of penicillin during the time of the acute thrombophlebitis and for a long period after all signs of the thrombophlebitis had subsided. Therefore, the use of large doses of penicillin was justified.

SUMMARY

1. Recovery in a case of thrombophlebitis of the cavernous sinus with multiple pulmonary abscesses complicating pregnancy is reported.

2. A brief discussion of the case history and of pertinent clinical and therapeutic items in regard to thrombophlebitis of the cavernous sinus is included.

Council on Pharmacy and Chemistry

REPORT OF THE COUNCIL

*The Council authorized the publication of a report [Report of the Council: Possible Dangers of Tetrathionate Therapy of Thromboangiitis Obliterans, J. A. M. A. **133**:693 (March 8) 1947] advising against the clinical use of sodium tetrathionate because of the potential dangers of nephrotoxic actions as revealed in animals. Dr. Frank V. Theis takes exception to this caution, believing that there is no danger with clinical doses. Dr. Geza de Takats also has protested against the report. The Council believes that these letters should be published. They are reproduced here.* AUSTIN SMITH, M.D., *Secretary.*

Supplemental Report on Sodium Tetrathionate

April 4, 1947

Dr. Austin Smith, Secretary
Council on Pharmacy and Chemistry
535 N. Dearborn Street
Chicago 10, Illinois

Dear Doctor Smith:

I am concerned by the action of your committee in officially advising, in the March 8 issue of THE JOURNAL, that the "use of Tetrathionate in medicine should be abandoned" because of its nephrotoxic effect. To abandon the use of Tetrathionate, on the "substantial evidence" you present, would deprive many patients suffering with thromboangiitis obliterans, thrombophlebitis and other related diseases of intravascular clotting from what are believed to be the benefits of its use. Your conclusion is based on the results of a limited study with relatively large doses of the drug given experimental animals. It overlooks completely the clinical investigations and successful use of Tetrathionate as a therapeutic agent. I know of no report of toxic effect from the clinical use of Tetrathionate.

In 1936 Freeland and I were responsible for having Tetrathionate (Searle) prepared and made commercially available. I have used or supervised the use of more than 15,000 ampules of Tetrathionate supplied to me by G. D. Searle and Company and have been satisfied with the results in treating more than 500 patients. Confirming our observations, de Takats (*Surgery* **14**:661 [Nov.] 1943) stated that the "inexpensive and nontoxic sodium tetrathionate is now in routine use on our (his) surgical service" and that "clinically there can be no doubt about the beneficial influence of intravenous sulfur compounds especially in migrating phlebitis"; and "in the acute stages of Buerger's disease the intravenous use of sulfur has given surprising results." Allen, Barker and Hines in their recent book state that to their knowledge "no untoward reactions have followed the administration of sodium tetrathionate." It may be that the Council, carrying the weight and responsibility that it does to the medical profession, will wish to review its opinion in the light of further evidence on the clinical investigations with sodium tetrathionate as well as interpret the results of animal studies with the therapeutic doses used in clinical medicine. These doses have not been toxic.

The nephrotoxic effects of Tetrathionate which are cited followed the administration of relatively huge doses to experimental animals. For the dog, Chen, Rose and Clows reported the lethal dose as being 1.0 mg. per kilogram body weight or 140

times the therapeutic clinical dose, while Gilman determined the dose to be 250 mg. per kilogram or 36 times the clinical dose. Gilman's statement that "Tetrathionate is toxic to the renal tubule to a degree that moderate doses produce complete anuria within less than an hour" may be questioned from the data he presented. The "moderate doses" he used were excessively large (i. e. 11.0 Gm. in a single injection to a dog produced anuria in seventy-five minutes) and were toxic doses compared to our recommended clinical dose of 0.4 or 0.6 Gm. for an adult weighing over 70 kilograms. His report showed that with 7 times the human dose given rabbits and 16 times the human dose given dogs, none of the animals died and there were no demonstrable toxic or ill effects. There are few systemic therapeutic drugs of value which have a greater factor of safety than Tetrathionate.

Prior to our clinical use and investigations of Tetrathionate, we too studied the toxic effects on rabbits. Series of rabbits were given daily intravenous doses of 0.1, 0.2, 0.4 and 0.6 Gm. of Tetrathionate, which were equivalent to 9, 18, 36 and 72 times respectively the clinical dose per kilogram recommended for therapeutic use. Daily injections of 0.4 Gm. (35 times the clinical dose) for three days (total of 1.2 Gm.) resulted in death of the animals from twelve hours to three days after the third dose. Autopsy study revealed edema and necrosis of the liver, kidney, heart and other organs. Rabbits given the 0.1 Gm. dose (9 times the clinical dose) daily for twelve days (total of 1.2 Gm.) showed no ill effects, thrived and gained weight. Autopsy study revealed no evidence of nephrotoxic or any other toxic effect. Studies on these animals included blood and platelet counts, hemoglobin, blood clotting time and glutathione, fibrinogen and prothrombin contents. Even with the fairly large doses used in the latter series of rabbits we found no deleterious effect.

The clinical dose which we recommended after extensive clinical and animal investigations with various size doses is 0.4 or 0.6 Gm. of sodium tetrathionate. This is equivalent to an average of 7 mg. per kilogram, a really small fraction of the toxic doses used in the investigations by Gilman. Even our most intensively treated patients show no evidence of nephrotoxic or other toxic effects; some of these have been followed for ten years and have received more than 300 injections. Occasionally, transient reactions with faintness, nausea, abdominal discomfort or weakness have followed an initial or too rapid injection. However, the same reactions occur with the use of sodium thiosulfate. Prompt relief is obtained by the ingestion of milk, food or water. Smaller subsequent doses or slower injection will almost always prevent recurrences of these reactions.

Our clinical investigation during the past ten years has included laboratory studies on the urine, blood cell counts, clotting time, sedimentation rate, basal metabolism rate and determination of the blood cholesterol, lipids, fatty acids, phospholipids, nonprotein nitrogen, urea, protein, creatinine, glutathione, sugar, calcium, potassium, sodium, prothrombin and oxygen and carbon dioxide capacity and content (saturation) of the arterial and venous blood. The results of some of these studies have been published and are available in the surgical literature (*Arch. Surg.* **38**:191 [Feb.] 1939; **40**:190 [Feb.] 1940; *Surgery* **11**:101 [Jan.] 1942; *Ann. Surg.* **113**:1107 [June] 1941). When changes followed injections of Tetrathionate they were usually favorable for the continued use of the drug; no deleterious effects have been observed.

My own analysis of Gilman's reported observations is that (1) Tetrathionate, when used in doses comparable to those used clinically, is not toxic to experimental animals and (2) the conversion and elimination of the Tetrathionate from the body are much slower than for the thiosulfate solution. However, the delay in eliminating the drug in the body is not sufficiently prolonged to produce deleterious or toxic cumulative effect from daily injections and is the reason for our original decision, ten years ago, for substituting the tetrathionate for the thiosulfate solution. We arrived at the same conclusion from the results of studies on the elimination of the sulfur in the urine. This part of the investigation was done with the cooperation of Dr. Andrew McNally, toxicologist, who originally suggested the use of Tetrathionate.

In view of the benefit which so many patients with thromboangiitis obliterans, thrombophlebitis and other diseases with tendency for intravascular clotting have received from the therapeutic use of Tetrathionate (Searle) and in the absence of any clinical or laboratory evidence of toxicity in the doses we advise, it would appear that the Council should review all the information available and reconsider its recommendation that the "use of Tetrathionate in medicine should be abandoned."

Yours truly,

FRANK V. THEIS.

Dr. Geza de Takats of Chicago also has written as follows under date of April 7, 1947:

I have delayed giving you my reaction to the statement made by your committee regarding tetrathione because I wanted to study the article of Gilman and also look over our records regarding any untoward results with the drug.

Obviously, sodium tetrathionate as used in Gilman's experiments was being used in a dose about thirty-five times the dose used in patients. I might say that our group has never made any renal function tests before and after use of sodium tetrathionate in man. However, with the exception of dizziness, flushing of the face or short attacks of nausea which may follow any intravenous injection, no harmful effects have been obtained.

My assistants and I use tetrathione rather sparingly and certainly have made no such extensive use of it as has Dr. Frank Theis and his group. However, if your committee based its opinion on the data available from Gilman's experiments, very few potent drugs can be used at present.

If you gave a dog or rabbit thirty-five times the dose of prostigmine, mecholyl, heparin, dicumarol and many others, there would be widespread toxic damage and mortality among the animals.

Certainly the quoted experiments do not reproduce clinical conditions and I am writing to suggest that the committee review its statement and if necessary conduct some more experiments based on controlled clinical material.

No nephrotoxic effects were observed in the patients treated by Dr. Theis and his associates and apparently these investigators feel that there is little if any danger of such effects from the doses that they have used clinically. However, when an agent is capable of interfering with the important but insufficiently known SH functions, a word of caution is not amiss. It should be realized at least that the dosage must be kept at a minimum and the renal function kept under close observation. Further observations may reveal additional valuable information.

Council on Foods and Nutrition

ACCEPTED FOODS

The following food, dehydrated tree-ripened flaked banana, used primarily in the feeding of infants and small children, has been accepted as conforming to the Rules of the Council on Foods and Nutrition of the American Medical Association.

JAMES R. WILSON, M.D., *Secretary.*

PREPARATIONS USED IN THE FEEDING OF INFANTS.

Kannenglesser & Company, New York

KANANA BANANA FLAKES.

Analysis (submitted by manufacturer).—Moisture 5.52%, ash 2.83%, fat (ether extract) 1.30%, protein (N × 6.25) 6.87%, carbohydrates, reducing 32.50%, sucrose 41.00%, starch 4.96%, total carbohydrates other than crude fiber (by difference) 83.48%.

Calories.—3.83 per gram; 109 per ounce.

Vitamins and Minerals.—	Per Hundred Grams
Vitamin A	154 U. S. P. units
Thiamine	0.199 mg.
Ascorbic acid	36.00 mg.
Riboflavin	0.318 mg.
Phosphorus	110.00 mg.
Magnesium	135.00 mg.
Sodium	406.00 mg.
Iron	2.70 mg.

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SATURDAY, JULY 26, 1947

RHEUMATIC FEVER CONTROL PROGRAM

The most comprehensive plan now in existence for control of rheumatic fever is probably that of the London County Council Rheumatic Scheme. School children in London are examined by school medical officers whose function is generally diagnostic. These doctors become especially interested and proficient in the recognition of rheumatic manifestations among the children routinely examined. General practitioners are invited to cooperate with the central office or one of its supervisory centers by referring to them children with diagnosed or suspected rheumatic fever who, in their opinion, should be admitted to a hospital. The London County Council has at its disposal 1,000 beds. When the central bureau passes on the case and the patient is approved by the medical referee the child is then transmitted by ambulance 20 miles to Carshalton or one of the other similar institutions in the country. These institutions, according to Parkinson,[1] are more than convalescent homes; they are probably best described as Hospital Schools. They have country surroundings with large open terraces alongside the wards onto which the beds can be easily run. It is the custom to carry out what is, in fact, open air treatment except in the most severe cases. Experienced medical officers believe that if the children are warmly clad there is no additional risk of infection or chill, that appetite is improved and sleep is sound. In severe carditis stress is placed on complete immobilization and ample nursing facilities. The children receive regular school instruction beginning with their convalescence from teachers appointed by the Ministry of Education. Parkinson stresses that education of rheumatic children is of great ultimate importance apart from the fact that the lessons help pass the time agreeably. The usual length of stay in one of these hospital schools is from six to twelve months.

The city of London has twenty supervisory centers distributed throughout, including the most thickly populated areas; ten of these are in connection with the outpatient departments of large hospitals. These centers carry out periodic supervision especially of patients discharged from the hospital centers, diagnose cases referred and instruct parents in child welfare. After school age young people with permanent valvular conditions are transferred to ordinary hospital outpatient clinics. Suitable employment is arranged for them by voluntary health societies such as the After-Care Association. The London City Council maintains a register of medical records and a card index of all the rheumatic children, past and present, who have been accepted at any center or hospital school. In 1938 the Council registered 22,800 children up to the age of 15 in London who were suffering or had suffered from rheumatic infection. This number represented an incidence of about 2.6 per cent of the child population.

With the passing of the New Health Act the whole of the health services and all the hospitals in the country will come under the control of the Ministry of Health in 1948; fourteen regions have been demarcated. They will be administered by regional councils, each working as far as possible from a main university center. The Royal College of Physicians appointed a rheumatic fever committee on which are represented the College, the Ministry of Health, the University of London, the Medical Research Council and the London County Council, with cardiologists and pediatricians concerned in the problem. This body is at present consultative, but it is believed that in the immediate future it will take the initiative in planning for a resumption of schemes like the London County Council scheme not only in London but in each of the regions into which the whole country has now been divided. The most urgent and the most difficult problem in connection with the control of rheumatic fever is the provision of sufficient beds for long term treatment of the rheumatic children.

In the United States public measures for the control of rheumatic fever are largely a matter of state and private concern rather than that of the governmental agencies. A good example of a state program is furnished by California. The program began with the formation in 1926 of the California Heart Association, the function of which is to arrange postgraduate education in heart disease for the physicians of the state. According to Rosenblum[2] the interest of physicians and of the general public was stimulated by the rheumatic fever program in Solano and Contra Costa counties. The program was supported by the state department of public health with funds made available by the Children's Bureau. Over 300 cases of rheumatic fever and rheumatic heart disease were detected and provision was made for hospital and convalescent care for many of them. Yearly symposiums open to all physicians in California and neighboring states were held in San Francisco, Los Angeles and San Diego

1. Parkinson, John: Public Measures for the Control of Rheumatic Fever in England, California Med. **66**: 230 (April) 1947.

2. Rosenblum, Harold: Rheumatic Fever Control Program in California, California Med. **66**: 233 (April) 1947.

under the sponsorship of community heart associations. These functions have been correlated by the California Heart Association and assisted by the medical schools and leading hospitals of the respective areas with teaching ward rounds, clinical demonstrations and lectures. In 1943 the California Tuberculosis and Health Association joined forces with the California Heart Association in forming a Heart Division of the California Tuberculosis and Health Association. This Heart Division provides an opportunity for health education and community organization in the field of rheumatic fever and rheumatic heart disease. Case registers for rheumatic children are being developed in San Francisco and Los Angeles counties. Although rheumatic fever and rheumatic heart disease are legally reportable in California, an adequate definition of the magnitude of the problem according to Rosenblum must await complete and effective reporting on a statewide basis. Here as well as in England the greatest shortcoming in the carrying out of the program is the lack of hospital and convalescent facilities. This shortcoming can be rectified by legislative action which will provide funds to assist local communities in developing programs and facilities.

THE PROBLEM OF AMEBIASIS

Albright and Gordon [1] point out that a considerable reservoir of unsuspected and unreported amebiasis has been brought back to the United States by the returning armed forces. The overall incidence of this type of infection in the United States is probably about 10 per cent. The so-called healthy carrier is a most important epidemiologic problem. Contrary to the widespread belief that Endameba histolytica may live and multiply within the lumen of the bowel without producing lesions or that an avirulent strain exists, they feel with Hargreaves [2] that the term "cyst carrier" is to be deprecated because every person who passes cysts must have vegetative amebas living on the tissues of his bowel wall and completing their normal development. Infection with the parasite always means invasion of the tissues with the production of pathologic lesions; cysts from asymptomatic carriers are fully pathogenic. Faust [3] demonstrated in dogs that there is no detectable difference in susceptibility to human strains of an acute, chronic or carrier type. The evidence concerning the inability of the parasite to live in the lumen without producing lesions is quite conclusive. Faust [4] in a series of 202 postmortem examinations conducted on accident cases in New Orleans found thirteen infections of E. histolytica demonstrated by the recovery of the organism in the bowel. The intestinal lesions consisted of pin point ulcerations, shallow crater-like lesions and rather extensive, superficial erosive processes of the mucosa. Amebas were demonstrated in each of these types of tissue involvement. These lesions represent the mildest type of damage to tissues reported in carriers and lend support to the belief that E. histolytica lives at the expense of the asymptomatic host. Albright and Gordon noted that amebic hepatitis develops in roughly one third of the patients who have never experienced diarrhea or dysentery. This strongly suggests that the "carrier" state is a pathologic condition warranting the same serious attention accorded other forms of the disease, since hepatitis is always secondary to invasive lesions of the wall of the bowel. The "carrier" represents an active stage of the disease, often completely free of symptoms, and for this reason the most easily overlooked and neglected. In this stage of the disease the opportunity is greatest for prevention of serious and even fatal complications.

Diagnosis of amebiasis is made by the case history, physical examination, proctoscopy, examinations of stools and only occasionally roentgenologic examination. Proctoscopy can provide invaluable information. Of special importance here are the color of the mucosa and the presence of bleeding points plus mucus, edematous areas and ulcerations. Early diagnosis of amebic hepatitis permits cure by medical means alone. The treatment includes a full course of emetine hydrochloride amounting to 0.65 Gm., with full courses of either carbarsone, chiniofon or diodoquin. Small abscesses will probably respond to this therapy, while larger abscesses require aspiration.

The aim of specific therapy is eradication of all parasites from the host. This is best accomplished by resorting both to emetine and to the enteric drugs, one having its action on parasites within the tissues and the other on parasites within the lumen of the bowel. Emetine hydrochloride given parenterally acts solely on trophozoites within the tissues. It leaves relatively untouched the trophozoites and cysts outside the tissues in the lumen or on the surface of the bowel. The enteric drugs, such as chiniofon, diodoquin, vioform and carbarsone, act on trophozoites and cysts of the second group within the lumen of the bowel. Albright and Gordon emphasize that the same therapeutic principles should be applied for all forms of amebiasis regardless of the phase of symptomatology. In Hargreaves' experience penicillin has proved life saving in severe refractory cases due to secondary bacterial infection. An attack on invading bacteria with penicillin and succinylsulfathiazole produced a dramatic improvement in these cases and made them more amenable to specific antiamebic treatment.

1. Albright, E. C., and Gordon, E. S.: Present Status of the Problem of Amebiasis, Arch. Int. Med. **79**: 253 (March) 1947.
2. Hargreaves, W. H.: The Treatment of Amebiasis, with Special Reference to Chronic Amebic Dysentery, Quart. J. Med. **15**: 1 (Jan.) 1946.
3. Faust, E. C.: Susceptibility, Resistance and Spontaneous Recovery in Dogs Experimentally Infected with Endameba Histolytica, Proc. Soc. Exper. Biol. & Med. **29**: 659, 1932.
4. Faust, E. C.: Amebiasis in the New Orleans Population as Revealed by Autopsy Examination of Accident Cases, Am. J. Trop. Med. **21**: 35 (Jan.) 1941.

Current Comment

EXPERIMENTAL INSOMNIA

Tyler reports [1] experiments on insomnia involving 600 subjects at the California Institute of Technology. Up to 112 hours of sleeplessness, no significant physiologic or biochemical changes were produced, except in the electrical activity of the brain. Little variation from the normal was noted in the blood sugar, hemoglobin, blood cell count, body weight and temperature. The excretion of total nitrogen and creatinine, ketosteroids, and the level of adrenal-like substances in the blood were little affected. The slight changes in blood pressure, respiration and heart rate that occurred could be attributed to the state of relaxation of the sleepy subjects. The psychologic disturbances were the most obvious changes that occurred. The alterations in behavior which were first noticed between thirty to sixty hours of sleeplessness were characterized by loss of memory, increased irritability, inattention and hallucinations or illusions. While in the majority of subjects these disturbances were mild, in some there were symptoms resembling those of acute schizophrenia. The only effects on psychomotor performance found were those that indicated reduced ability to maintain sustained effort and performance.

NEW EPIDEMIOLOGIC PROBLEMS

In discussing epidemiologic problems arising from human action in an instructive lecture titled "Epidemiology Today," Professor Burnet's [1] first example is infectious jaundice or hepatitis associated with the vaccination of American troops against yellow fever using vaccine containing small amounts of normal human serum. Infectious jaundice has appeared also after the use of syringes in the injection of penicillin and other medicines without thorough sterilization of the syringes between injections. The virus of one type of infectious jaundice may be in the blood of symptomless persons, and it can survive various procedures. Burnet's second example "of large-scale epidemiologic happenings produced by human action that was designed to prevent disease" is the development of strains of pathogenic organisms, particularly streptococci, which resist the action of sulfonamides and other drugs. In 1944 the recruits in many U. S. naval training camps were given sulfadiazine daily for some months in order to prevent streptococcic infections. The results were not satisfactory, as an unduly large number of such infections occurred. Two streptococcus strains (types 17 and 19) had acquired resistance to the action of sulfadiazine and infected persons who were receiving sulfadiazine for prophylaxis just as readily as virulent normal strains caused infection in unprotected persons. Fortunately the sulfonamide resistant strains remained sensitive to penicillin. Burnet likens the appearance of a drug resistant bacterial strain to the emergence of a new animal species. The events in naval training camps warn against subjecting a community or other large group to long continued chemoprophylaxis. Burnet also warns against dangers in far flung treatment of gonorrhea with a single drug. New and drug resistant strains of gonococci may become dominant, as in fact is illustrated by the sulfonamide resistant gonorrhea.

1. Presented by David B. Tyler before the Federation of American Society for Experimental Biology and abstracted in the Federation Proceedings, part-II, p. 218, March 1947.

1. Burnet, F. M.: Epidemiology Today, M. J. Australia **2**: 825 (Dec. 14) 1946.

THE FIRST ARTIFICIAL SPRING LEG

Teele [1] comments historically on the accomplishments of two ingenious citizens of Milton, Mass., one Mr. Benjamin Crehore, the other Capt. Lewis Vose. In 1798 Crehore began to manufacture bass viols, the first made in this country. In 1800 he made the first pianoforte manufactured in this country. His ingenuity was further challenged by the appearance in Milton of Dean Weymouth, a young man who had lost a leg in the service of his country. Crehore conceived the idea of making a wooden leg for him with joints in the knee, the ankle and the foot. After many modifications he succeeded in fitting out an artificial limb which worked easily. Capt. Lewis Vose, a saddler, arranged and put in the straps to secure the leg in place and the padding to make it work with comfort. History further relates that Mr. Weymouth used the artificial limb for a long time but, failing to obtain the money to pay for it, was obliged to return it to Mr. Crehore. Subsequently the leg disappeared from where it had been deposited and its fate is unknown. This artificial limb was believed to be the first of its kind made in this country. To confirm the claim to priority of invention, a statement of the facts in the case has been inserted in the Boston *Transcript*. A year has elapsed without response; apparently the claim to priority holds good.

MONGOLISM AND INFECTIONS IN PREGNANCY

Mongolism is a congenital but not hereditary general maldevelopment the cause of which is not known. The discovery by Gregg and others in Australia that maternal rubella in pregnancy may be followed by congenital cataract as well as cardiac and other malformations in the child led Ingalls and Davies [1] to look into the question of intercurrent maternal infections in pregnancies which produced mongoloid children. In a preliminary note they record 7 such cases, one each of influenza, rubella, mumps, mastoiditis, pleurisy, "grippe" and otitis media with sinusitis. The fact that in 6 of the 7 cases the maternal infection occurred at about the second month of pregnancy is, as they say, "provocative." This note should stimulate physicians to furnish data to determine whether there is a definite correlation between maternal infection in pregnancy and mongolism. Whatever the outcome of such analysis may be, the relations of maternal rubella to malformations in the fetus demand that everything possible be done to exclude maternal infections in pregnancy.

1. Teele, A. K.: The History of Milton, Mass., 1640 to 1887, Boston, Press of Rockwell and Churchill, 1887.

1. Ingalls, J. H., and Davies, J. A. V.: Mongolism Following Intercurrent Infectious Disease, New England J. Med. **236**: 437, 1947.

ORGANIZATION SECTION

Official Notes

GROUPS OF CLINICIANS TO AID SICK IN U. S. POSSESSIONS

Secretary of the Interior Krug recently requested the American Medical Association to appoint an advisory committee to select teams of competent clinicians to go for a short period to such places as Alaska, the Western Indian reservations, Puerto Rico and the Virgin Islands to aid the local physicians in the diagnosis and treatment of difficult cases of illness and to conduct clinics for the indigent with the view to provide a high grade of medical service. The advisory committee appointed comprises the Executive Committee of the Board of Trustees of the American Medical Association, Dr. George F. Lull, Secretary of the Association, Dr. Morris Fishbein, Editor of THE JOURNAL, and Dr. W. A. Morgan of Washington, D. C.

The first group, a Chicago team, departed from Minneapolis July 19 for three weeks of this service in Alaska. This team is composed of Drs. Jack Silverstein, Arthur Bernstein, George Holmes, Harry E. Barnett and John C. Fields, all from the Cook County Hospital in Chicago. The Alaskans will select their difficult cases in advance and assemble them in hospitals in order to avoid long trips and to save the time of the Chicago clinicians.

In accordance with Secretary Krug's request it is expected that another group of clinicians will be sent to Puerto Rico and the Virgin Islands in December for the purpose of aiding in difficult cases and of improving health conditions in those islands. The groups of clinicians selected will also work with public health officers, where they are available, in order to aid in bringing the best medical care to the underprivileged groups. Clinicians will be selected later from other medical centers to visit the Western Indian reservations. As this plan is expected to be continued, more clinical teams will be sent out next year.

Washington Letter

(From a Special Correspondent)

July 23, 1947.

U. S. Mission to Combat Health Problems in Greece

Three U. S. Public Health service officers were assigned to the American Economic Aid Mission to Greece to help that nation combat tuberculosis, typhoid, dysentery, hookworm, infant mortality and other medical problems. After a first hand study they may request assistance of other American medical workers. A health service spokesman said the trio, whose names were not announced, would help direct expenditure of the $3,000,000 public health fund requested from Congress. The Greek death rate from tuberculosis is reported to be between 250 and 450 per hundred thousand of population as compared with 40 in the United States. Tremendous sanitation problems resulted from destruction of 23 per cent of all dwellings in Greece. It was also announced that Dr. R. R. Sayres, director of the Federal Bureau of Mines, has resigned to rejoin the U. S. Public Health Service staff.

House and Senate Conference Studies National Science Foundation Bills

A Senate-House conference is ironing out differences in bills passed by both sides of Congress for establishment of a twenty-four member National Science Foundation to promote fundamental research for health and national defense. The group would supervise scientific activities for the government and promote "pure science." The House approved its bill by voice vote last week, and recently the Senate approved a similar measure by a vote of 79 to 8. The legislation, backed by President Truman and leading scientists, would set up a program costing between $20,000,000 and $30,000,000 annually. The group could not engage in atomic energy research without approval of the Atomic Energy Commission. The House bill would direct the foundation to formulate national policy for promotion of fundamental research and education in sciences, support research related to national defense, grant scholarships and graduate fellowships in medical, biologic and other sciences, foster interchange of scientific information in the United States and foreign countries, and establish special commissions on cancer research, on heart and circulatory diseases and on poliomyelitis and other degenerative diseases. Foundation members would be appointed by the President subject to Senate approval.

Board Appointed to Select Students for Study Abroad

President Truman did not appoint a medical person to the ten man Board of Foreign Scholarships set up to select American students for study abroad under the Fulbright act. The State Department has received about 12,000 applications, of which 5,000 are from veterans having preference under law. The law authorizes agreements with foreign governments to use part of moneys paid for American war surplus materials to finance studies of Americans in foreign educational institutions and for travel of foreign students to study in this country. Russia is not now eligible but may qualify under lend-lease settlement. Countries eligible are Australia, Austria, Belgium, Czechoslovakia, Denmark, Egypt, Finland, France, Greece, Hungary, Iran, Italy, the Netherlands, the Netherlands East Indies, New Zealand, Poland, Siam, Turkey, the United Kingdom, Burma, the Philippines and China.

Pan American Congress of Pediatrics to Meet in Mexico City in 1949

The Pan American Congress for Pediatrics selected Mexico City for the second congress, to be held in 1949. More than 600 delegates and guests attended the closing sessions here. Dr. Felix Hurtado of Cuba was chairman. The chairman of arrangements was Dr. Edgar P. Copeland; Dr. Harry A. Ong, was assistant chairman.

Coming Medical Meetings

Alaska Territorial Medical Association, Fairbanks, July 28-30. Dr. William J. Blanton, Juneau, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., The Homestead, Sept. 4-6. Dr. James R. Bloss, 418 Eleventh St., Huntington 1, W. Va., Secretary.

American College of Surgeons, New York, Waldorf-Astoria Hotel, Sept. 8-12. Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secretary.

American Congress of Physical Medicine, Minneapolis, Hotel Radisson, Sept. 2-6. Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secretary.

American Congress on Obstetrics and Gynecology, St. Louis, Municipal Auditorium, Sept. 8-12. Mr. Karl S. Richardson, 24 West Ohio St., Chicago, Business Manager.

American Roentgen Ray Society, Atlantic City, Sept. 16-19. Dr. H. Dabney Kerr, University Hospitals, Iowa City, Secretary.

Colorado State Medical Society, Denver, Shirley-Savoy Hotel, Sept. 17-20. Mr. Harvey T. Sethman, 1612 Tremont Place, Denver 2, Executive Secretary.

National Medical Association, Los Angeles, Aug. 18-22. Dr. John T. Givens, 1108 Church Street, Norfolk 10, Va., Secretary.

Nevada State Medical Association, Reno, Sept. 4-6. Dr. Moreton J. Thorpe, 17 N. Virginia St., Reno, Secretary.

Oregon State Medical Society, Portland, Sept. 4-6. Dr. Thomas S. Saunders, 1020 S. W. Taylor St., Portland 5, Secretary.

Pennsylvania, Medical Society of the State of, Pittsburgh, Hotel William Penn, Sept. 15-18. Dr. Walter F. Donaldson, 500 Penn Avenue, Pittsburgh 22, Secretary.

Southeastern Dermatological Association, Knoxville, Tenn., Hotel Andrew Johnson, Aug. 31. Dr. A. H. Lancaster, 608 W. Main Ave., Knoxville, Secretary.

Utah State Medical Association, Salt Lake City, Sept. 11-13. Dr. Ray T. Woolsey, 610 McIntyre Bldg., Salt Lake City 1, Secretary.

Western New York and Ontario Urological Society, Saranac, N. Y., Saranac Inn, Sept. 3-6. Dr. Albert M. Crance, 525 S. Main St., Geneva, N. Y., Secretary.

Bureau of Information

LOCATION OF PHYSICIANS

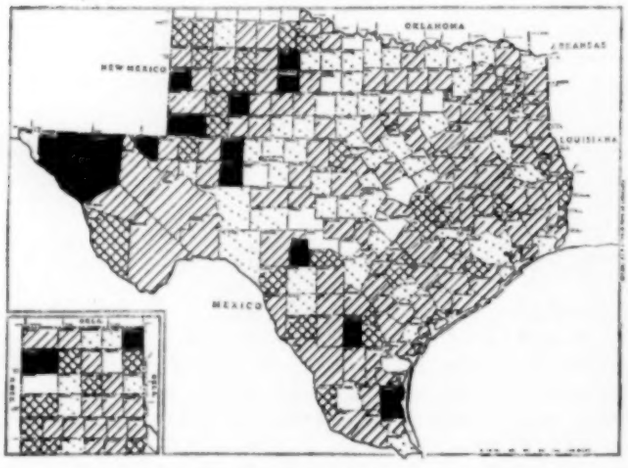
Virginia Shuler
Chicago

This is the sixth of a series of charts showing the trend in locations of physicians from April 1946 to January 1947.

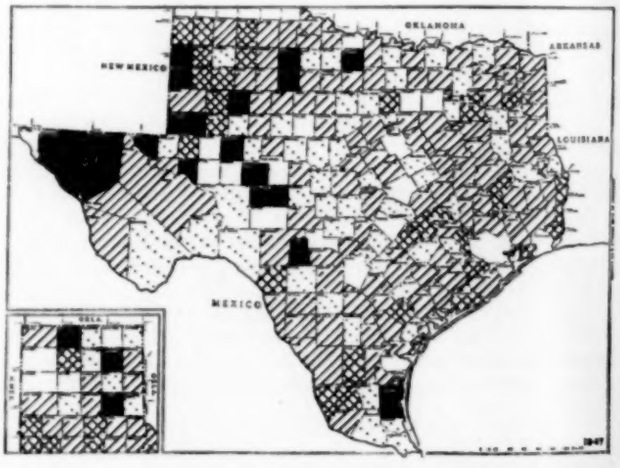
"Conditions in Texas are still somewhat abnormal. During the past year we have been successful in a good many instances in locating doctors in certain communities. Nevertheless, we still have a rather extensive list of areas where doctors are needed. We are doing our best to get this information into the hands of doctors seeking locations, including Texas physicians, as well as physicians in other states. Efforts are being made by the Association, in cooperation with the three medical colleges in Texas, to impress all graduates with the need for physicians in rural areas and the advantages offered by such areas. There are no areas in the state at this time where the need for physicians is really critical. In most areas there is at least one physician within a radius of 25 miles. That has always been true in Western and Southwestern Texas, which is largely sparsely settled ranching country. Migration of physicians during the past year has shown a trend of movement from rural communities to the cities. The rural communities, however, could benefit greatly if money available under the Federal Hospital Construction Act were to be used in the construction of needed hospitals and health centers. Texas has 254 counties and 129 local medical societies. Every county in Texas is embraced in some medical society. At present there are forty or fifty small towns, communities and villages in Texas that have requested physicians. Not all of these requests represent real need, but most of them do. We keep in close touch with the proper officials and committees of county medical societies as to the need for doctors in their respective jurisdictions. We do not always list applications filed with us by drug stores, chambers of commerce, civic clubs, and so forth, or even doctors. We carefully check each claim and list only the deserving."

Dr. Holman Taylor, Secretary,
State Medical Association of Texas,
1404 West El Paso Street, Fort Worth 3.

TEXAS—1946



TEXAS—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

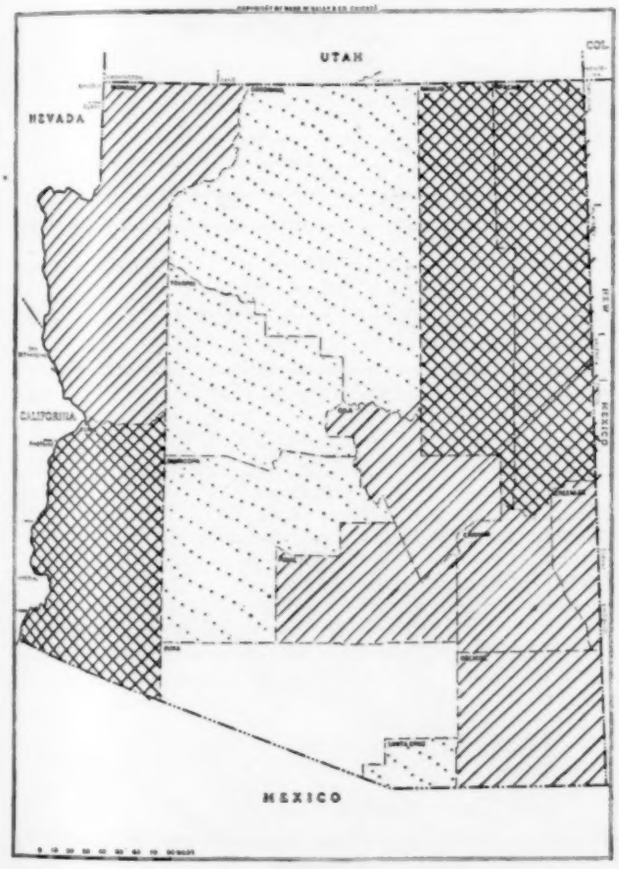
Areas in Texas Requesting General Practitioners

County	Town	Population
Austin	Wallis	815
Burleson		13,940
Burnet	Burnet	1,945
	Marble Falls	2,500
Cass	Avinger	624
Clay		9,488
	Henrietta	2,391
Cooke		24,909
Crane		2,841
Culberson		1,320
	Van Horn	1,130
Dallam	Dalhart	4,682
Denton	Lewisville	1,123
	Pilot Point	6,000
Donley		1,187
	Clarendon	3,000
Eastland	Eastland	3,849
Edwards		2,075
Gonzales		26,075
Gregg	Gladewater (Negro physician)	4,454
Hall		...
	Memphis	3,869
	Turkey	930
Hamilton		13,303
Hansford	Spearman	1,200-1,400
Harris	Seabrook	862
Harrison		...
Hemphill	Canadian	2,151
Henderson		31,822
Hill	Blum	441
	Hillsboro	7,759
	Itasca	1,759
	Malone	429
Houston	Crockett (Negro physician)	4,536
Jim Wells	Alice	7,732
Karnes	Runge	1,001
Kaufman	Kemp	1,000
	Terrell (Negro physician)	10,481
Kenedy		700
Kinney		1,301
Lamar	Paris	18,678
Lee	Dime Box	300
	Lexington	531
Leon		17,733
McLennan	Crawford	471
	McGregor	2,002
Menard		4,521
Montague	Nocona	2,605
	Saint Jo	1,010
Newton		13,700
	Newton	1,400
Raine	Emory	886
Real	Camp Wood	778
	Leakey	500
Reeves		9,423
	Pecos	4,853

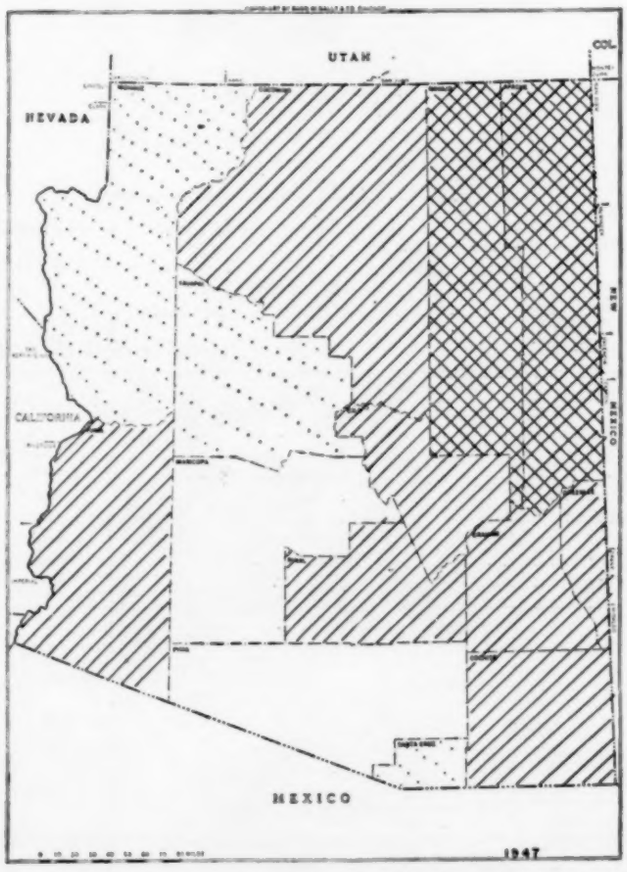
Areas in Texas Requesting General Practitioners—Continued

County	Town	Population
Runnells	Ballinger	4,472
Sabine		10,896
San Augustine		12,471
San Patricio	Portland	907
San Patricio and Aransas	Aransas Pass	4,095
Shelby		29,235
Smith	Lindale	820
	Troup	1,526
Somerville	Glen Rose	1,050
Terrell		2,388
Tipscomb	Higgins	741
Titus	Mount Pleasant	4,528
	Talco	912
Tyler	Woodville	1,500
Upshur	Gilmer	3,138
Walker	Huntsville	5,108
Ward		10,121
	Monahans	3,944
Winkler		4,878
	Kermit	2,584
	Wink	1,954
Wise	Bridgeport	1,735
	Decatur	2,578
Zavala	Crystal City	6,529

ARIZONA—1946



ARIZONA—1947



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Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Area in Arizona Requesting General Practitioner

County	Town	Population
Gila	Payson	487

"Arizona is a state of great geographic distances, yet there are but fourteen counties in the vast expanse of the state. Each county maintains a county medical society, hence its populace is assured medical care. Several of the counties have heavy Indian populations and a scattered white population of lesser numbers. Two of the counties, Maricopa and Pima, account for better than two thirds of the white population of the state, Phoenix and Tucson (Maricopa and Pima counties respectively) being the two cities in which the population is centralized. Aside from these two cities there are few other towns equaling or topping 15,000 in population.

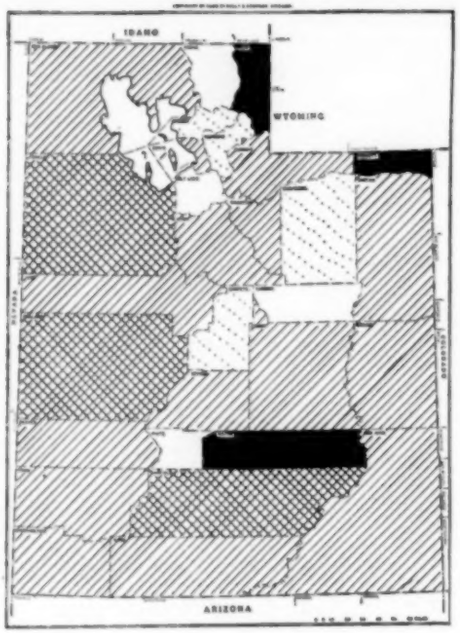
"All communities in the state supporting hospitals are presently well supplied with physicians and surgeons. The outlying districts needing and desiring more medical personnel will first need to set up hospitals and local facilities for the care of their sick.

"On the whole, Arizona has a full complement of doctors of medicine when facilities for caring for the sick of the state are fully considered. Specialists in practically every field of practice, and certified specialists, are fully represented in this complement of physicians and surgeons.

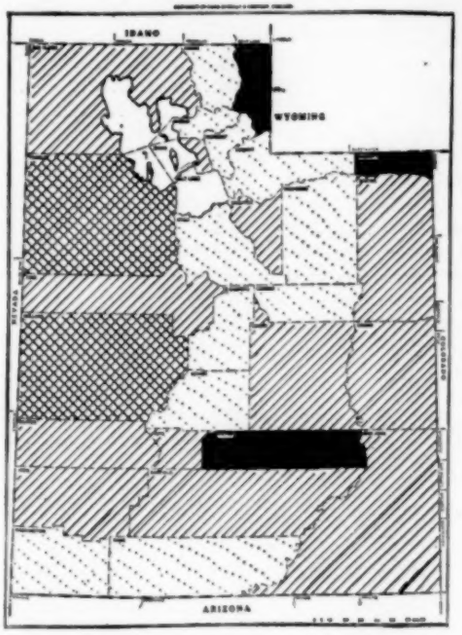
"As the white population in the more remote areas of the state increases and as hospital facilities become available in these areas, the spread of high class physicians and surgeons in such communities may be anticipated."

DR. FRANK J. MILLOY, Secretary,
The Arizona State Medical Association,
423 Heard Building, Phoenix.

UTAH—1946



UTAH—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

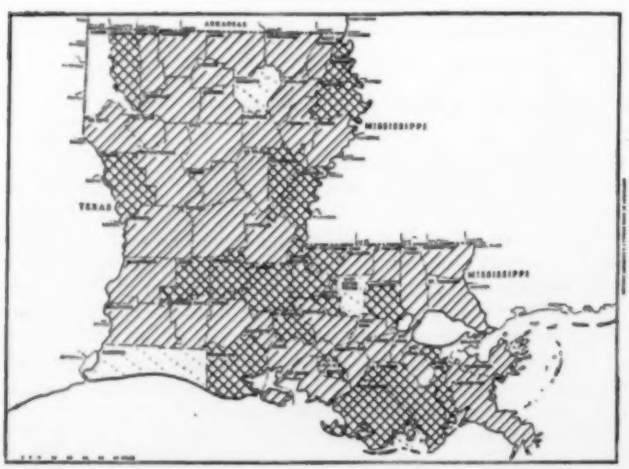
Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in Utah Requesting General Practitioners

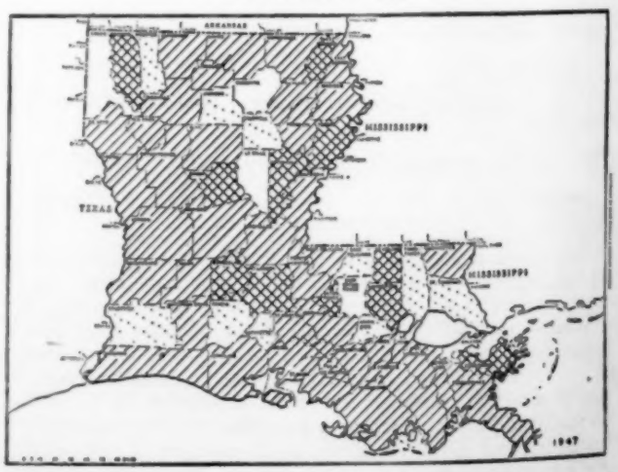
County	Town	Population	County	Town	Population
Beaver	Milford	1,393	Salt Lake		211,623
Duchesne		8,958	San Juan		4,712
	Duchesne	907	Tooele		9,138
Juat		7,392		Tad Park	2,000
Millard		9,613	Uintah		9,898
	Delta	1,304	Wasatch		5,754
Rich	Randolph	500		Kamas	1,500
Summit		8,714	Wayne	Loa	500

Mr. W. H. Tibbals, Executive Secretary,
Utah State Medical Association,
68 S. Main Street, Salt Lake City.

LOUISIANA—1947



LOUISIANA—1946



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by parish.

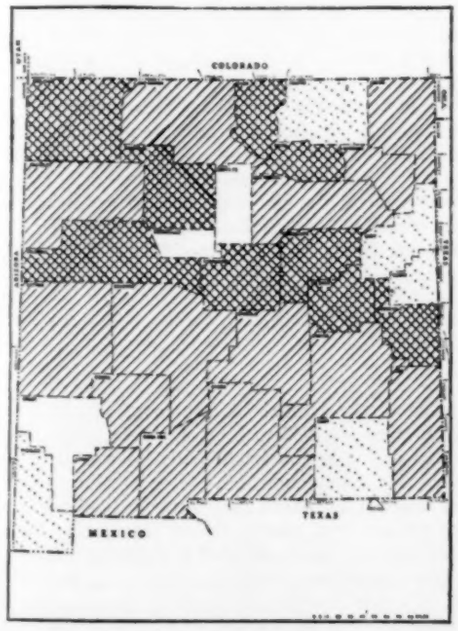
Key: Physician-population ratio by parish: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in Louisiana Requesting General Practitioners

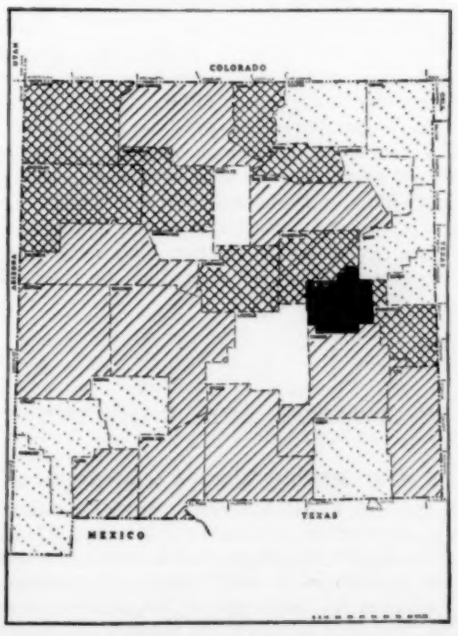
Parish	Town	Population
Acadia	Church Point	1,802
Allen	Oakdale	3,033
Avoyelles	Cotton Port	1,196
	Moreauville	815
Beauregard	Morryville	1,216
Bienville	.....	23,933
Caddo	Belcher	300
	Oil City	1,447
Caldwell	Columbia	947
Catahoula	.....	14,618
Claiborne	.....	29,855
	Homer	3,497
DeSota	Mansfield	4,065
East Carroll	Lake Providence	3,711
East Feliciana	Jackson	5,384
Franklin	Winnsboro	2,834
	Wisner	617
Grant	Colfax	1,354
Iberville	Carville	250
	Maringouin	708
Lafayette	Lafayette	19,210
LaSalle	Urania	1,016
Lincoln	Choudrant	438
	Dubach	749
	Ruston	7,107
Madison	Tallulah	5,712
Morehouse	Oak Ridge	373
Natchitoches	.....	40,997
Plaquemines	Buras	1,183
	Burrwood	400
Red River	Coushatta	1,280
Sabine	Many	1,474
	Pleasant Hill	737
St. Charles	Norco	1,529
St. John the Baptist	Edgard	268
	Reserve	3,000
St. Landry	.....	71,481
	Eunice	5,254
	Opelousas	8,980
	Port Barre	929
	Washington	1,264
St. Mary	Franklin	4,274
St. Tammany	Covington	4,123
Tangipahoa	Ponchatoula	4,001
Union	.....	18,203
Vernon	.....	19,142
	Leesville	2,829
Webster	Cotton Valley	1,199

DR. P. T. TALBOT, Secretary,
Louisiana State Medical Society,
1430 Tulane Avenue, New Orleans.

NEW MEXICO—1946



NEW MEXICO—1947



Population, based on 1943 estimated Census figures, per active physician (excludes teaching, research, hospital administration, public health and state hospitals) by county.

Key: Physician-population ratio by county: plain area 1-999, dotted area 1,000-1,499, striped area 1,500-2,999, checked area 3,000 and over, solid black area no doctor.

Areas in New Mexico Requesting General Practitioners

County	Town	Population
Guadalupe	Santa Rosa	2,310
Quay	San Jon	650
	Tucumcari	6,194
Roosevelt	Elida	330
San Miguel	.....	27,910
Taos	.....	18,528

"In general, I should say that the cities of 5,000 or more in population are well supplied with physicians at the present time, with the possible exception of a few specialists, such as nose and throat men, which are needed in certain areas. In the rural areas there are still several localities which are entirely without any physician. One could conservatively estimate that there are at least a dozen areas in the rural sections—towns of about 1,000 population—where good general practitioners are needed."

DR. H. L. JANUARY, Secretary,
New Mexico Medical Society,
Albuquerque.

GOVERNMENT SERVICES

ARMY

ARMY AWARDS AND COMMENDATIONS

Colonel Derrick T. Vail

The Legion of Merit was recently awarded to Dr. Derrick T. Vail, now professor of ophthalmology at Northwestern University Medical School, Chicago. The presentation was made June 3 by Dr. Paul R. Hawley, medical director of the Veterans Administration, for outstanding services rendered in the European Theater of Operations from Oct. 5, 1942 to Oct. 14, 1944, when Colonel Vail served as senior consultant in ophthalmology at ETO headquarters. The citation read in part as follows: In addition to establishing and maintaining a singularly high standard for the treatment and supervision of ophthalmologic cases, Colonel Vail demonstrated exceptional qualities of adaptability and a thorough professional knowledge by encouraging the adoption of many improvements, resulting in the conservation of time and manpower. His whole hearted interest in the rehabilitation and care of blinded American soldiers, his keen foresight and exceptional devotion to duty reflect the highest credit on himself and on the armed forces of the United States. Dr. Vail graduated from Harvard Medical School, Boston, in 1923 and entered the military service in September 1942.

Colonel William D. Graham

The Legion of Merit has been awarded to Col. William Donald Graham, M. C., U. S. Army. The citation read in part as follows: For performing exceptionally meritorious services as commanding officer of the 158th General Hospital in England from November 1944 to July 1945. He was responsible for the outstanding accomplishments of the hospital in the field of neurosurgery and plastic surgery during a period of maximum flow of casualties. His work has reflected great credit on the Medical Department of the United States Army. Dr. Graham graduated from the University of Minnesota Medical School in 1930 and entered military service in 1934.

Colonel L. Holmes Ginn Jr.

The United States of America Typhus Commission Medal has been awarded to Col. L. Holmes Ginn Jr., M. C., U. S. Army. The citation read as follows: He performed meritorious service in connection with the work of the United States of America Typhus Commission during the period May 8 to July 10, 1945 in the Rhine Province Military District, Germany. As surgeon of the Fifteenth United States Army he exhibited keen foresight and rare judgment in planning and directing effective typhus fever control measures throughout the army area. Colonel Ginn has also been awarded the Legion of Merit and the Bronze Star Medal with the Oak Leaf Cluster. Colonel Ginn graduated from the Medical College of Virginia, Richmond, in 1927 and entered the military service in July 1927.

Major Arthur R. Hummel

The Legion of Merit was recently awarded to Major A. R. Hummel, M. C., A. U. S., now of Detroit, for exceptionally meritorious conduct in the performance of outstanding services in the Middle East from January 1943 to November 1945. Dr. Hummel graduated from Wayne University College of Medicine in Detroit in 1941 and entered the service June 12, 1942.

Dr. Herald R. Cox

The United States of America Typhus Commission Medal has been awarded to Dr. Herald R. Cox of Pearl River, N. Y. The citation read as follows: Dr. Herald R. Cox, civilian, distinguished himself as a consultant to the United States of America Typhus Commission from December 1942 to December 1945. While continuing his notable work with the development of typhus vaccine, he also gave valuable aid in producing serums to treat diseases of the typhus group and in investigating the problems of a vaccine against scrub typhus. His work was highly beneficial in the campaign against typhus fever and was a distinct contribution to scientific knowledge. Dr. Cox graduated from the School of Hygiene, Johns Hopkins University, in 1931.

NAVY

NAVY AWARDS AND COMMENDATIONS

Rear Admiral Charles S. Stephenson

The Legion of Merit has been awarded to Rear Admiral Charles S. Stephenson (MC), U.S.N., retired, of Washington, D. C. The citation read in part as follows: For exceptionally meritorious conduct in the performance of outstanding services as medical officer in charge of the division of preventive medicine in the Bureau of Medicine and Surgery and as navy medical representative on the National Research Council from Sept. 8, 1939 to Nov. 24, 1942. Displaying outstanding organizational ability and farsightedness in making recommendations concerning the preservation of health, he was in large measure responsible for safeguarding and maintaining the excellent physical condition of Navy, Marine Corps and Coast Guard personnel during World War II. He contributed to the success of the National Research Council in the execution of its vital war mission. His professional skill, initiative and tireless devotion to duty reflect the highest credit on himself and the United States Naval Service. Dr. Stephenson graduated from the Medical School of Vanderbilt University in 1912 and entered the naval service in 1913.

Captain Frederick R. Hook

The Navy Commendation Ribbon has been awarded to Capt. Frederick R. Hook (MC), U.S.N. The citation read as follows: For outstanding performance of duty as medical officer in command of the United States Naval Hospital at Oakland, Calif., from Sept. 1, 1942 to Nov. 15, 1943. By his outstanding professional and organizational skills he was in large measure responsible for completion of the expansion program which made his hospital the third largest naval hospital in the nation. Through his untiring efforts during this period he contributed to the efficient treatment rendered to thousands of war casualties and to the expeditious return to duty of a majority of these patients. Captain Hook's fulfilment of his exacting assignment was in keeping with the highest traditions of the United States Naval Service. Dr. Hook graduated from the University Medical College of Kansas City in 1913 and entered the military service in November 1917.

Captain James R. Thomas

The Navy Commendation Ribbon has been awarded to Capt. James R. Thomas (MC), U.S.N. The citation read in part as follows: For outstanding performance of duty as medical officer in command of the United States Naval Mobile Hospital Number One from May 2, 1942 to Oct. 31, 1943; and subsequently as medical officer in command of the United States Naval Hospital, Corpus Christi, Texas, from Nov. 10, 1943 to Aug. 1, 1945. By his constant devotion to the exacting details of his assignments, he contributed materially to the war effort and upheld the highest traditions of the U. S. Naval Service. Dr. Thomas graduated from the University of Illinois College of Medicine in 1917 and entered the naval service in September 1917.

Lieutenant Commander Charles R. Gennaria

The Navy Commendation Ribbon has been awarded to Lieut. Comdr. Charles R. Gennaria (MC), U.S.N.R., of Shamokin, Pa., for excellent service as senior medical officer aboard the U. S. S. *Leedstown* from Jan. 31 to Oct. 20, 1944, during the amphibious assaults and capture of Kwajalein, Guam, Peleliu and Leyte. During this period he organized and directed the activities of his department, which was taxed far beyond its normal capacity by the casualties from enemy action. That an exceptionally low death rate was achieved despite the fact that many of the seriously wounded were long delayed in reaching the ship is recognized in the citation as affording testimony to his outstanding ability and devotion to duty. His conduct and courage were at all times in keeping with the highest traditions of the United States Naval Service. Dr. Gennaria graduated from Jefferson Medical School in 1923 and entered the naval service Sept. 24, 1942.

PUBLIC HEALTH SERVICE

DR. SPENCER RESIGNS AS CHIEF

Dr. R. R. Spencer has resigned as chief of the National Cancer Institute of the U. S. Public Health Service, Washington, D. C., and Dr. Leonard A. Scheele, assistant chief of the institute, has been appointed chief. Dr. Spencer's resignation does not mean that he will withdraw from the work of the institute. He desired to be relieved of the increasing load of administrative duties in order to spend full time on "two phases of the cancer program that interest him most—professional education and research." In his new capacity he will direct the institute's expanding program for professional training of young physicians in the diagnosis and treatment of cancer and at the same time carry on research on the mechanism of cellular and species survival, which is related to the cancer process. Dr. Spencer is widely known also in the field of research for work in association with R. R. Parker in developing a vaccine against Rocky Mountain spotted fever.

Dr. Scheele has been in the Public Health Service since 1933 and was a member of the staff of the institute for several years before the war. During the war he served in Italy in Military Government and Allied Commission operations and later was in charge of the preventive medicine section of a division of supreme headquarters, Allied Expeditionary Force in Northwest Europe. He was also a representative of the medical section of the Allied Control Council in its initial operations in Berlin after Germany surrendered. He has been awarded the American Typhus Commission Medal and the Legion of Merit.

DR. IVY EXECUTIVE DIRECTOR OF ADVISORY COUNCIL

Dr. A. C. Ivy, Chicago, vice president of the University of Illinois, has been appointed executive director of the National Cancer Advisory Council to succeed Dr. George M. Smith, professor emeritus of Yale University, who resigned for reasons of health. Dr. Ivy, who was for many years head of the Department of Physiology at Northwestern University Medical School, has been a member of the council for two years. He will take up the part time post as executive director of the council on his return from Germany late in July.

VETERANS ADMINISTRATION

EDUCATIONAL COURSES FOR DISABLED VETERANS

About two hundred complete educational courses, ranging from high school subjects to automobile mechanics, are available to ill and disabled veterans in Veterans Administration hospitals. They are offered to patients as part of their educational therapy activities. The courses cover, in addition to academic high school material, a variety of prevocational and vocational technical subjects such as poultry raising, radio repair, photography and plastics. Commercial subjects include shorthand, typing, bookkeeping and problems of operating a small business. The two hundred courses available are listed in a catalogue distributed to all 126 Veterans Administration hospitals and homes.

One armed patients can learn to type in special classes organized by the Veterans Administration educational retraining sections. Despite their handicaps, these patients use standard typewriter keyboards in the course.

AWARD TO DR. HAWLEY

Dr. Paul R. Hawley, chief medical director of the Veterans Administration, has been awarded the Gorgas Award for 1947, which will be presented to him in Boston at the annual meeting of the Association of Military Surgeons, November 13-15. This award is given annually by the executive council of the Association of Military Surgeons for some notable contribution in the field of military medicine. It is sponsored by Wyeth, Incorporated, Philadelphia.

HOSPITAL IN MONTANA

The army engineers at Fort Peck, Montana, have opened the bids received for the construction of the 100 bed veterans' hospital at Miles City. The lowest bid was $4,437,000, which was 32 per cent above the government estimate of the cost of construction, and so no award has as yet been made.

PERSONALS

Brig. Gen. Guy B. Denit, who is on duty in the Surgeon General's Office, received the honorary degree of Doctor of Science from the Medical College of Virginia, Richmond, June 17.

Dr. Julian J. Keller has been assigned chief surgeon of the veterans' hospital at Tuscaloosa, Ala. Dr. Keller, who has been in the Veterans Administration service for many years and during World War II served in the medical corps of the Army, recently was on the staff of the veterans' hospital at Bay Pines, Fla.

Dr. Cyril H. Francis, Columbus, Ohio, has been appointed medical director for the Veterans Administration in Ohio, Michigan and Kentucky. Dr. Francis has been serving as chief of professional services with the Veterans Administration. He served in the navy medical corps during the war and previous to that time was in private practice in Los Angeles.

Dr. George T. McMahan has been assigned as manager of the Veterans Administration hospital at Gulfport, Miss., succeeding Dr. G. T. Sheffield, who has been assigned at his own request as psychiatrist to the Veterans Administration center at Biloxi.

Dr. Robert C. Cook, deputy director of the Veterans Administration department of medicine and surgery, represented the Veterans Administration at the International Congress of Military Medicine and Pharmacy in Switzerland; June 2-9. Dr. Cook is a graduate of Rush Medical College in Chicago and has been with the Veterans Administration since 1919.

Dr. Arthur Leiter has been appointed general medical examiner in Indiana by the Veterans Administration and will be located in Indianapolis. Dr. Leiter formerly practiced in Columbia City, Ind., and was health commissioner of Whitley County.

The plan to assign Dr. Cameron S. Guild to the branch office in San Francisco (THE JOURNAL, May 24, p. 384) was changed and he has been assigned to the branch office in New York City.

PHYSICIANS SEPARATED FROM SERVICE

ARMY MEDICAL CORPS OFFICERS RECOMMENDED FOR/OR RELIEVED FROM ACTIVE DUTY

Alabama

Bashinsky, Leo M.....Birmingham
Bograd, Nathan.....Montgomery
Bouchelle, McLemore.....Birmingham
Britton, James W.....Anniston
Conwell, Donald P.....Jasper
Culbreth, George G.....Andalusia
Fackler, William B. Jr.....Wadley
Graves, Robert S.....Gadsden
Greenlees, David L.....Greensboro
Horne, Cecil P.....Montgomery
McCorkle, Walter W.....Gadsden
McVay, Leon V., Jr.....Mobile
Merrill, Byrd F.....Dothan
Nettles, Thomas E.....Tunnel Springs
Rollins, Stacy L. Jr.....Atmore
Timberlake, Gideon B.....Stevenson
Underwood, Ralph O.....Phil Campbell

Arkansas

Calaway, Robert L.....Little Rock
Costley, Lawson C. Jr.....Pine Bluff
Gladden, Jean C.....Harrison
Kerr, Robert T.....Little Rock
Vandergriff, Harris T.....Paragould
Wade, Henry K., Jr.....Hot Springs
Watson, John E.....Little Rock

California

Anderson, Milford X.....Los Angeles
Babcock, Kenward O.....Ontario
Beard, Crowell.....San Mateo
Bode, Arnold G. H.....Sierra Madre
Bouland, Thomas G. Jr.....Glendale
Bridwell, Willard S.....Glendale
Brizzolara, Louis G.....San Francisco
D'Elia, Alphonse E.....Wilmar
deSilva, Paul L.....Oakland
Dolkas, Constantine P.....Alhambra
Dutra, Frank R.....Sacramento
Faust, Joseph M.....Los Angeles
Finkle, Eugene D.....Oakland
Graham, William H.....Crescent City
Green, Robert L.....Los Angeles
Griffin, Herschel E.....San Francisco
Hepplewhite, James G.....Chico
Hibbard, Lester T.....Los Angeles
Holladay, William T.....Long Beach
Hopkins, Robert E.....Lodi
Jeans, Howard S.....Pasadena
Juul, Clement O.....Oakland
Kane, Carl L.....Glendale
Klein, Thomas.....Madera
Lambert, Edward R.....Santa Barbara
Magg, Davin.....Hollywood
McNiel, Ralph W.....Alhambra
O'Connor, John J.....San Francisco
Robinson, Harold H. Jr.....Sacramento
Rosenberger, Maurice D.....Los Angeles
Rosenburg, Marvin J.....Los Angeles
Salomon, Maurice S.....San Francisco
Schwartz, Monte.....San Francisco
Schwartzman, Herman.....Belden
Scott, Robert F.....Los Angeles
Shipounoff, George C.....San Francisco
Stadtner, David A.....San Francisco
Steinbach, Owen K.....Los Angeles
Taketa, Tom.....Tulelake
Thomas, Robert C.....Vallejo
Uyeyama, Kahn.....Berkeley
Van Deventer, William C.....Long Beach
Van Scoyoc, James G. Jr.....Los Angeles
Vaughan, Robert A.....Los Angeles
Weeden, William F.....Alameda
White, Manuel.....Berkeley
Wickett, William H. Jr.....Fullerton
Wilson, William H.....Redlands
Yates, Charles D.....Modesto

Colorado

Burnett, Berry H.....Englewood
Dunklee, George M.....Denver
Hall, Robert F.....Grand Junction
Lombardi, James C.....Denver
Marasco, Paul B.....Grand Junction
Midkiff, Layton D.....Denver
Miller, William C.....Trinidad
Twombly, George C. Jr.....Fort Morgan
Vanden Bosch, Marvin P.....Denver
White, Stanley M.....Denver
Wurtzebach, Lorenz R.....Denver

Connecticut

Beloff, Jerome S.....Meriden
Brooks, Wilbur S.....East Haddam
Freeman, Joseph.....New London
Haley, John C.....Mt. Carmel
Jenkins, Ward S.....Danbury
Moser, David W.....Rocky Hill
Reidy, Joseph C.....Winsted
Spellman, Francis A.....New Haven

Delaware

Hall, Samuel W. III.....Dover
Keyser, Morton.....Wilmington
Stillman, Eugene H.....Wilmington

District of Columbia

Corey, Merle I.....Washington
Dowd, James F.....Washington
Evans, Peyton R. Jr.....Washington
Greenberg, Arnold S.....Washington
Greenlee, Charles E.....Washington
Hensel, Albert E. Jr.....Washington
Johnsen, David S.....Washington
Weller, George L. Jr.....Washington

Florida

Breinin, Goodwin M.....Miami Beach
Corso, Vincent P.....Miami
Erwin, Charles R.....Miami
Grimes, George R.....West Palm Beach
Henschen, Hal.....Oakland
Petteway, Charles H.....Lakeland
Wilson, Charles F.....Orlando
Wing, Breckinridge W.....Winter Park

Georgia

Brannen, Edmund A.....Atlanta
Brown, Charles E.....Barnesville
Clary, Thomas L. Jr.....Augusta
Deen, Oliver F. Jr.....Douglas
Fisher, Albert Jr.....Macon
Galloway, William H.....Atlanta
Griffith, Joseph E.....Adairsville
Jordan, Willis P. Jr.....Columbus
Kelly, Gordon McN.....Augusta
McMillan, Eugene C. Jr.....Atlanta
Palmer, Clarence B.....Covington
Ross, Thomas L.....Macon
Smith, Reuben E.....Lawrenceville
Wood, David L.....Dalton

Idaho

Farber, Jack R.....Payette
Paull, Don R.....St. Anthony
Sutton, Richard P.....Oakley
Taylor, Eugene E.....Moscow

Illinois

Barrett, Charles V. Jr.....Chicago
Briskin, Harold L.....Chicago
Brown, William H.....Chicago
Byers, Richard S.....Granite City
Coombs, James W.....LaGrange
Curtiss, Paul H. Jr.....Harvey
Delicate, William E.....Edwardsville

Illinois—Continued

Douglass, Thomas C.....Chicago
Eissler, Kurt R.....Chicago
Fish, William D.....Evanston
Freier, Morton L.....Chicago
Greene, Warren W.....Skokie
Greenwood, Benjamin S.....Champaign
Haeberlin, John B. Jr.....Chicago
Henke, Charles P.....Chicago
Henry, Nelson W.....Chicago
Hofrichter, Frank C.....Chicago
Icenogle, Grover D.....Chicago
Irwin, George E. Jr.....Kankakee
Kaemerle, Harold K.....Chicago
Kendall, Hillis E.....Villa Grove
Kruglik, Meyer.....Chicago
Lambert, Claude N.....Chicago
Leibovitz, Martin.....Chicago
Maguire, Roderick H.....Chicago
Marzano, Vincent V.....Chicago
Miller, Jacob M.....Chicago
Moore, Stanley W.....Vandalia
Murfin, Walter W.....Decatur
Prempas, Thomas G.....Chicago
Rappaport, Henry.....Chicago
Rose, Gabriel.....Kankakee
Rosenberg, Edward F.....Chicago
Rosenberg, Sidney.....Chicago
Rosenthal, Sol R.....Chicago
Rosi, Alcide L.....Chicago
Schwerin, George S.....Chicago
Schweitzer, Albert W.....Lexington
Scott, Thomas G.....Dwight
Sellett, Andrew J.....Peru
Shaffer, William T.....Chicago
Sklar, Isidore A.....Chicago
Soffer, Joseph I.....Chicago
Stone, Bernard M.....Chicago
Strauch, Franz A.....Chicago
Streicher, Daniel L.....Chicago
Tarnoff, Joseph F.....Chicago
Tomlinson, William A.....Springfield
Vandenberg, Roy E.....Dolton
Wechsler, Jacob I.....Chicago
Weiland, Albert S.....Chicago
Weisberg, Seymour W.....Chicago
Werth, Joseph A.....Waterloo
White, Michael S.....Chicago
Winter, Tyrus D.....Belleville

Indiana

Bethea, Robert O., Jr.....Madison
Brown, David B.....Gary
Downard, Leland F.....Liberty
Eaton, Merrill T. Jr.....Bloomington
Ettl, Edward J.....South Bend
Foxworthy, Donald T.....Madison
Garrett, Robert A.....Indianapolis
Gregg, Edwin E.....Indianapolis
Hanson, Mark C.....Indianapolis
Headlee, Charles R.....Indianapolis
Hodurski, Zigfield.....Indianapolis
Kuhn, Frederick L.....Plymouth
Lovett, Harvey D.....Zionsville
Ludwick, Harry A.....South Bend
Malstaff, Comiel M.....South Bend
McClellan, John B.....Muncie
Mellen, John R.....Indianapolis
Price, Ronald D.....Indianapolis
Randak, Edward F. Jr.....Crawfordsville
Sandy, William A.....Indianapolis
Sheller, Tom G.....Akron
Siebenmorgen, Paul.....Terre Haute
Symmes, Alfred T.....Indianapolis
Thom, Jay W.....Gosport
Van Ness, William C.....Summitville
Westfall, George S.....Indianapolis
Wisenbaugh, Paul E.....North Liberty

PHYSICIANS SEPARATED FROM SERVICE

Iowa

Beebe, Kenneth P.....Wever
Blair, Donald W.....Dubuque
Brown, David R., Jr.....Sioux City
Butterfield, Elwyn T.....Jefferson
Dye, William S., Jr.....Centerville
Fitzpatrick, Matthew R.....Mason City
Foley, Walter E. Jr.....Davenport
Fox, Robert T.....Hubbard
Hicks, Murwyn L.....Iowa City
Kersten, Paul M.....Fort Dodge
Kessler, Donald L.....Cedar Rapids
Olson, Ranald E.....Montezuma
Smiley, George W.....Grinnell

Kansas

Abbuehl, Don R.....Atchison
Knosp, Norman C.....St. Francis
Merriam, Wallace.....Kansas City
Meyer, Paul W.....Kansas City
Miller, Ted W.....Arkansas City
Swan, Major M.....Erie
Vander Velde, Stanley L.....Emporia

Kentucky

Bisig, Charles J.....Louisville
Gilliam, Melvin R.....Danville
Haynes, Philip E.....Hopkinsville
Krupp, Abraham W.....Louisville
Lowry, Conie C.....Princeton
McCabe, Robert J.....Covington
Quertermous, John C.....Salem
Rosenbaum, Orvin S.....Louisville
Roy, William A.....Lexington
Walker, John W., Jr.....Irvine
Wygal, James M.....Corbin

Louisiana

Appleby, Homer Q.....Slaughter
Bethea, Hardee.....New Orleans
Davidoff, Theodore.....DeRidder
Dreyfus, Albert E.....New Orleans
Finkelstein, Julius.....New Orleans
Landry, Louis V.....Port Allen
Leake, William S. Jr.....New Orleans
Mayer, Louis Jr.....Baton Rouge
Mellinger, George T.....New Orleans
Mintz, Stanley R.....Monroe
Ragan, Charles J.....Pineville
Robichaux, Richard E.....Raceland
Sellers, Harry W.....Alexandria
Swindle, James T.....New Orleans
Tyler, Thomas B.....New Orleans
Zoller, Harry.....New Orleans

Maine

Herrick, Carleton S. Jr....South Brewer
Oestrich, Alfred.....Mexico
Robertson, Frank O. Jr.....Bethel

Maryland

Blumenthal, Leonard E.....Baltimore
Bondurant, Herbert W.....Chevy Chase
Buckey, Robert B.....Baltimore
Dann, Alfred H. .....Baltimore
Genecin, Abraham.....Baltimore
Haigley, Thomas B.....Baltimore
Johnson, Robert W. III.....Baltimore
Kaiser, Irwin H.....Baltimore
Kemper, Charles A.....Baltimore
Kleiman, Bernard S.....Baltimore
Muse, William T.....Baltimore
Parker, Robert T.....Towson
Pearce, William F.....Baltimore
Polek, Melvin F.....Baltimore
Rinehart, Arthur M.....Baltimore
Royer, Earl L.....Woodlawn
Spier, Andrew A.....Cumberland
Taylor, Irving J.....Ellicott City
Varner, Philip H.....Chevy Chase

Massachusetts

Ambadgis, Theodore T.....New Bedford
Armitage, Henry G. Jr.....Haverhill
Bell, John F.....Boston
Chansky, Daniel.....Dorchester

Massachusetts—Continued

Bonner, Charles D.....New Bedford
Croeker, Augustus T.....Boston
Crocker, Seth C.....Milton
Dobelle, Martin.....Pittsfield
Fine, Michael B.....Dorchester
Flaherty, James R.....Worcester
Grokoest, Albert W.....Norwood
Guterman, Bert.....Worcester
Hunter, Richard E.....Worcester
Hurley, Joseph P.....Worcester
Kirby, Francis A., Jr.....Whitman
Lake, Fredric D.....Springfield
Liberman, Charles.....Winthrop
Magno, Henry A.....Malden
Malone, Edward H.....Jamaica Plain
Maloney, John M.....Springfield
Oppenheim, Harry.....Dorchester
Shea, Daniel J. Jr.....Chestnut Hill
Spence, Philip S. Jr.....Boston
Sullivan, Richard J.....North Brookfield
Suzor, Lester G.....Chicopee
Tavares, John M.....Somerville
Turner, Harland G.....Boston
Velonis, Stamatis G.....Marblehead
Weinstein, Abraham.....Roxbury
Wigglesworth, William C.....Harvard

Michigan

Bird, Harrie W. Jr.....Bloomfield Hills
Burton, Irving F.....Detroit
Byberg, Robert A.....Detroit
Clark, William P.....Hudson
Haeck, William.....Grand Rapids
Hall, Edward J.....Hartford
Hayner, Russell A.....Howell
Jackson, William F.....Rogers City
Jones, Melvin C.....Bay City
Lovell, Barney K.....Detroit
Milgrom, Sidney.....Detroit
Mulder, Gerrit A.....Grand Rapids
Phillipson, Chester.....Flint
Rosenbaum, Herbert.....Detroit
Roth, Emil M.....Grand Rapids
Ruhl, Frank G. Jr.....Detroit
Sorensen, Maurice G.....Detroit
Thompson, John R. Jr.....Tecumseh
Usndek, Harold E.....Detroit
Van Solkema, Arthur.....Grandville
Wallaert, Albert A. J.....Detroit

Minnesota

Boardman, Dalmon V.....Twin Valley
Carlander, Lester W. Jr.....Minneapolis
Coughlan, Vernon H.....Minneapolis
Countryman, Frank W.....St. Paul
Davis, Thayer C.....Wadena
Estrem, Ralph L.....Fergus Falls
Firestone, George M.....St. Paul
Folsom, Bartlett L.....Littlefork
Gillam, John S.....Minneapolis
Hays, John C.....St. Paul
Hoganson, Donald E.....Bemidji
Hultgen, William J.....St. Paul
Johnson, Donald W.....Fairmont
Kantar, Bruce L.....Minneapolis
Kusske, Douglas R.....New Ulm
Levin, Louis.....Rochester
Merrick, Robert L.....Minneapolis
Simmonds, Harry N.....St. Paul
Struxness, Erling B.....Belgrade
Turbak, Charles E.....Canby
Wallace, George T.....Rochester
Walstad, Paul M.....Minneapolis
Watson, Robert McL.....St. Paul

Mississippi

Lobrano, Charles M.....Centerville
Santa Cruz, Edgar W.....Gulfport

Missouri

Barnett, Julian H.....St. Louis
Boles, Clifford R.....University City
Brookreson, Alton F.....Poplar Bluff
Cross, Glen O.....St. Louis

Missouri—Continued

Doyle, Joseph P. Jr.....East Prairie
Haberer, Justin J.....St. Louis
Halperin, Phillip H.....Kansas City
Hamtil, Edward W.....St. Louis
Hibbard, Blaine Z.....Kansas City
Kromer, Charles J.....St. Louis
Lipsitz, Ellis S.....St. Louis
Loeb, Virgil Jr.....St. Louis
McFadden, James F., Jr.....Clayton
Murphy, John W., Jr.....Kirkwood
Roberts, Richard S.....Trenton
Sawhill, Fred A.....St. Louis
Simmons, Leroy.....Sarcoxie
Stipp, Charles G.....St. Louis
Swenson, Alvin L.....Independence
Treharne, Frank E.....Independence
Van Ravenswaay, Arie C.....Boonville

Montana

Lewis, Rand C.....Bozeman
Rossiter, Henry D.....Sheridan

Nebraska

Aita, John A.....Omaha
Bednar, Bryce R.....Omaha
Browne, Kenneth M.....Omaha
George, John H.....Omaha
Glenn, Elmer E.....Omaha
Gogela, Louis J.....Omaha
Larson, Frank C.....Peru
Merrick, Robert B.....Alliance
Sprague, Lavern D.....Omaha
Stuart, Robert L.....Lexington
Wagner, William F.....Omaha
Watson, James W.....Grand Island
Yohe, Charles D.....Omaha

New Hampshire

Buber, Luther W.....Berlin
Matthews, Thomas V.....Concord

New Jersey

Bertland, Alexander U.....Flemington
Borsher, Irving P.....Bloomfield
Boysen, Otto T.....Egg Harbor City
Colonna, Joseph A.....West New York
Cooper, Jehu P. III.....Metuchen
Cox, John R.....Mullica Hill
D'Amato, Charles R.....E. Rutherford
Day, Harold E.....Phillipsburg
Deitz, Joseph R.....Trenton
Della Penna, Samuel J.....Haskell
DePietro, Vincent P. .....Union City
Dern, Samuel M.....Perth Amboy
DeTroia, Frederick C. .....Newark
Einhorn, Harvey P.....Newark
Esgro, Philip.....Glassboro
Gilbert, Philip D.....Camden
Goodman, Stanley J.....Jersey City
Greenfield, Sylvan J.....Newark
Harris, Morris.....Bloomfield
Jurewicz, Stanley T.....Paterson
Kaplan, Samuel D.....Hillside
Krakower, Alvin H.....Paterson
Levine, Edward P.....Newark
Lioy, Anthony D.....Grantwood
Mansfield, Richard.....West New York
Marcussen, Robert M.....Maplewood
Mason, Daniel.....Jersey City
McCarthy, Joseph C.....Spring Lake
Midura, Peter P.....Perth Amboy
Mulliken, David K.....Leonia
Murphy, John J.....Jersey City
Nelson, Clifford H.....Irvington
Rich, Robert E.....Newark
Sandford, Francis R.....Arlington
Sasso, Albert.....Belleville
Shapiro, Albert B.....Vineland
Tabankin, Alvin.....Newark
Titus, Norman E.....Red Bank
Van Gorder, Charles O.....Westwood
Wein, Sidney F.....Passaic
Weinberg, Clarence R.....Newark
Whitaker, Henry J.....Pitman

PHYSICIANS SEPARATED FROM SERVICE

New Mexico

Daviet, Leslie L.....La Cruces
Keeler, Claude C.....Fort Bayard
Lane, Russell C.....Silver City
Richardson, George S.....Roswell

New York

Ackerman, Martin.....New York
Azulay, Abraham.....New York
Berger, Arnold L.....Bronx
Berman, Louis.....Brooklyn
Bochetto, Joseph F., Jr.....Brooklyn
Bonner, William F.....Batavia
Breed, Ernest S.....Lyndonville
Brock, Henry J.....Buffalo
Brown, Charles A.....Bronx
Brown, Prescott L.....Albany
Burnett, James J.....Binghamton
Burns, Thomas A.....Syracuse
Caltabiano, Carmelo G.....Brooklyn
Campion, William F. X.....Bronx
Carreras, Miguel A.....New York
Carter, Robert E.....Jamaica
Catapano, John P.....Brooklyn
Cerasano, Ernest A.....Schenectady
Cherico, Pasquale.....New York
Clark, George W.....Chazy
Colangelo, S. J.....North Collins
Cotlove, Ernest.....New York
Davids, Arthur M.....New York
DeAngelis, Carmelo E...Jackson Heights
Delterzo, Julius R. .....Brooklyn
Demuth, Edwin L.....New York
DeRespinis, Benedict J. .....New Dorp
DeRostaing, Caesar.....Oceanside, L. I.
Dodge, Henry W. Jr.....Bronxville
Dorsey, William F.....Long Island City
Effron, Samuel A.....New York
Evans, Richard D.....Utica
Fahey, Thomas J. Jr.....Syracuse
Falussy, Anthony G.....Elmhurst
Feffer, Henry L.....Brooklyn
Feldman, Harold J.....Buffalo
Fonseca, Juan E.....New York
Forsyth, Henry H. Jr.....Rochester
Frishwasser, Edward J.....Bronx
Frost, Frank T.....Oswego
Gardner, Murray H.....Watertown
Geary, John R. Jr.....Lockport
Glasser, Seymour.....Brooklyn
Glick, Benjamin W.....New York
Goldfischer, Murry.....New York
Grabicki, Zygmunt P.....Schenectady
Greene, William F.....New York
Greenfield, Leigh S.....Rochester
Gregorius, Frederick J. C.....New York
Grishman, Arthur.....New York
Hacker, Arthur J.....Rensselaer
Haggerty, John J.....New York
Heffernan, John A.....Rochester
Herzfeld, Melvin G.....New York
Ierardi, Peter A. Jr.....Brooklyn
Karusiatis, Vincent V.....Brooklyn
Kassander, Paul.....New York
Kaufman, Irving C.....Brooklyn
Khuri, Naim N.....Binghampton
King, Samuel E.....New York
Klingon, Gerald H.....Brooklyn
Klugler, Joseph.....Bronx
Kolodny, Maxwell H.....New York
Kozinn, Philip J.....Brooklyn
Leone, John A.....Niagara Falls
Levine, Daniel I.....Hempstead, L. I.
Lindsay, William W.....Freeport
Long, Frederick R. Jr.....Woodhaven
Lukash, Leslie I.....New York
Madey, Stephen L.....Buffalo
Marchetta, Frank C.....Utica
Matlin, Paul.....Brooklyn
McKinstry, George C.....Webster
McMillan, George J.....Oakfield
Meyer, Albert H.....Brooklyn

New York—Continued

Meiselas, Leonard E.....Brooklyn
Meister, Edward H. Jr....East Setauket
Micelotta, Joseph D.....Forest Hills
Miller, Howard B.....New York
Miller, Saul D.....New York
Moore, Robert J.....Albany
Moreland, Randall B.....New York
Morelli, Henry E.....Bronx
Novich, Emanuel A.....Yonkers
O'Brien, Matthew J.....Buffalo
Olmsted, Burton L.....Buffalo
O'Neil, Charles H. Jr.....Gloversville
Osher, Alexander P.....Long Island City
Petronella, Horace G....Jackson Heights
Piccione, Gary A.....Rockville Centre
Polansky, Marvin.....Albany
Posner, Leonard.....Brooklyn
Pugh, David F.....New York
Rabourn, William O.....Port Byron
Radin, Samuel D.....Bronx
Rapp, Bergen A.....Hempstead
Rekers, Paul E.....Douglaston, L. I.
Robins, Edwin.....Brooklyn
Romberg, George H.....White Plains
Root, Douglas L.....Albany
Rosenfeld, Daniel M.....Brooklyn
Rosmarin, Samuel.....Brooklyn
Rossomando, Alfred H.....New York
Roth, Daniel.....New York
Roth, Emanuel.....Brooklyn
Rothman, Martin.....Brooklyn
Russell, William F. Jr.....New York
Rutherford, George L.....Albany
Sauer, Paul K.....New York
Schaye, Alvin A.....New York
Schlein, William.....Brooklyn
Siegel, Jacob.....Bronx
Silvis, George M. Jr.....Brooklyn
Sirkin, Jacob.....Newark
Solomon, Samuel.....Brooklyn
Spevak, Jack.....Brooklyn
Stache, Ernest G.....Astoria, L. I.
Stark, Jesse D.....New York
Steiner, Karl (Ignaz).....New York
Strade, Henry A.....New York
Strauss, Elias.....New York
Sullivan, James M.....New York
Sullivan, Norbert P.....New York
Tillim, Sidney J.....Amityville
Upjohn, Richard H.....Brooklyn
Upshur, Alfred P.....New York
Van Buren, John J.....Brooklyn
Van Graafeiland, Willard W...Rochester
Varmus, Frank.....Freeport, L. I.
Warres, Herbert L.....Brooklyn
Warshaw, Arthur H.....New York
Weiner, David O.....Brooklyn
Weisberg, Howard K.....New York
Wetherell, Frederick E.....Syracuse
Weitz, Martin L.....Laurelton
Zanghetti, Gene J.....New York
Zolondek, Sol.....Brooklyn

North Carolina

Barnwell, Howard B.....Edneyville
Beale, Jefferson D. Jr.....Winston-Salem
Boone, Henry.....Jackson
Boyette, Dan P. Jr.....Ahoskie
Brantley, Julian T.....Greensboro
Cann, John E.....Greensboro
Codington, Herbert A., Jr....Wilmington
Collett, James R.....Morganton
Dimmette, Robert M.....Ellerbe
Featherston, William P.....Durham
Fisher, George W. Jr.....Elizabethtown
Hubbard, Robert T.....Spray
Kester, John M.....Wilmington
Lafferty, John O.....Charlotte
McDowell, Harold C.....Cherryville
Moore, James LeG.....Raleigh
Mubarak, Jameel S.....Morresville
Noble, Baxter G.....Kinston

North Carolina—Continued

Powell, Jess A. Jr.....Edenton
Pressly, Claude L.....Statesville
Ross, Thomas W.....Charlotte
Rothman, Joseph.....Bessemer City
Valk, Henry L.....Winston-Salem
Wetmore, Robert J.....Durham
Youngblood, Vernon H.....Concord

North Dakota

Brandsted, Ernest C.....Amenia
Brimi, Robert J.....Cooperstown
Donovan, Edward J.....Langdon
Hauser, William P.....Dickinson
Rothnem, Morris S.....Fargo
Mitchell, George H.....Milnor
Wood, Robert A.....Jamestown

Ohio

Adler, James S.....Amanda
Black, Louis A.....Kenton
Brogden, William E.....Canton
Brown, Delatus E.....Columbus
Brown, Edward S.....Shaker Heights
Ceraldi, Bernard A.....Cleveland
Damashek, Samuel.....Dayton
Etzkorn, Peter A.....Delphos
Fishell, Richard D.....Findlay
Hendershot, Robert G.....Wingett Run
Hughes, Owen F.....Bowling Green
Hummel, George P.....Cleveland
Kelty, Richard H.....Canton
Kimerer, Neil B.....Toledo
Lemmon, James F.....Akron
Lewis, Robert N.....St. Clairsville
Manning, Thomas L., Jr.....Cleveland
Miller, Louis.....Cleveland Heights
Miller, Stanley H.....Cincinnati
Mohler, George T.....Coshocton
Munns, Thomas A.....Oxford
Nilges, Richard G.....Fairview Village
Otis, John.....Lakewood
Prugh, Dane G.....Dayton
Quinn, Thomas W.....Napoleon
Randt, Clark T.....Lakewood
Ross, Paul S.....Columbus
Schwinger, Aaron.....Cincinnati
Segal, Abraham J.....Cleveland Heights
Somsel, Clarence P.....Dayton
Styblo, Clarence J.....Cleveland
Voskamp Ramirez, Jack..Cleveland Hgts.

Oklahoma

Bugg, Robert N.....Oklahoma City
Copeland, Evan L.....Oklahoma City
Curtis, Selvie J.....Mustang
Davis, William O. .....Cushing
Forrest, Herbert J.....Tulsa
McClellan, James T.....Claremore
Neel, Roy L.....Oklahoma City
Oxley, William N.....Wilson
Rector, William L. Jr.....Ardmore
Sapper, H. V. L. Jr.....Oklahoma City
Sanders, Harold R.....Oklahoma City
Strode, Jack W.....Stillwater
Tolbert, Jack B.....Oklahoma City
Turner, Edwin C.....Oklahoma City

Oregon

Dobbin, Harold W.....Portland
Griswold, Herbert E. Jr.....Portland
Lee, George F.....Union
Molenkamp, Delmar J.....Portland
Royce, Byron F.....Salem
Skirving, John H.....The Dalles
Smith, John C.....Hood River

Pennsylvania

Auer, Edward T.....Philadelphia
Barness, Lewis A.....Warrington
Barone, Samuel F.....Pittsburgh
Bishop, Paul H. Jr.....Bethlehem
Blizard, Richard W.....Philadelphia
Blumenthal, Charles.....Philadelphia

PHYSICIANS SEPARATED FROM SERVICE

Pennsylvania—Continued

Bolton, Howard A.....Aspinwall
Carideo, Louis N.....Drexel Hill
Castagna, Armand.....Philadelphia
Cohen, Sheldon G.....Pittston
Dattner, Herman B.....Wilkes Barre
DeMarco, Anthony R. .....Erie
DeSantes, Frank A. .....Altoona
Dix, Robert C., Jr.....Nicholson
Donaldson, David H. Jr.....Cannonsburg
Erlichman, Irving F.....Philadelphia
Fortnum, Walter G.....Eddington
Fox, John L.....Upper Darby
Gartland, John J. Jr.....Stonehurst
Gibbs, Robert L.....Barnesboro
Goracci, Armando F.....Old Forge
Gratch, Michael.....Carbondale
Hosay, John J.....Dickson City
Hough, Charles E.....Lehighton
Howanitz, Emil.....Glen Lyon
Huber, Gerard N.....Latrobe
Johnson, William S.....Philadelphia
Klimas, Enoch G. Jr.....Philadelphia
Knox, Frederick H. Jr.....Philadelphia
Kramen, Samuel V.....Philadelphia
Lepper, Norman.....Rockledge
Lipcius, Frank.....Lester
Mercurio, Frank.....Downingtown
Merriman, Wilson C.....Beaver Falls
Mervine, Thomas B.....Philadelphia
Meyers, John L.....Shillington
Miller, Edgar A.....Gettysburg
Miller, Henry N.....Reading
Miller, Richard E.....Tamaqua
Mills, William L.....Philadelphia
Milstead, Laurence C.....Allentown
Mitro, William M.....Donora
Owen, John J.....Media
Parker, Andrew J.....Pittsburgh
Pashuck, Eugene T.....Philadelphia
Rigby, Charles E. Jr.....Folsom
Rogers, Hugh J.....Bellefonte
Roos, Leon.....Juniata, Altoona
Rushmore, Charles H.....Clark's Greens
Salus, Karl.....Philadelphia
Scarpa, Harry C.....Philadelphia
Schaaf, William J.....Mt. Oliver
Smith, Charles M.....Pittsburgh
Sullivan, Herbert H.....Pittsburgh
Sutton, Robert C.....Mifflintown
Swanson, Elmer R.....Harwick
Toland, Joseph J. III.....Philadelphia
Wagner, Henry S.....Cressona
Warshafsky, Morton.....Philadelphia
Witmer, Donald B.....New Holland
Yankevitch, John J.....Freeland
Zellis, Allan.....Philadelphia

Rhode Island

Bigelow, Nolton H.....Providence
Bojar, Samuel.....Providence
Gordon, Calvin M.....Providence

South Carolina

Eaddy, Albert M.....Columbia
Thomas, John D. Jr.....Loris
Thomason, James R.....Fountain Inn
Weston, Christian T., Jr.....Charleston

South Dakota

Brookman, Bruce T.....Vermillion
Crowley, James H.....Rapid City
Mitchell, Charles B.....Pierre
Shaw, Millard R.....Tripp

Tennessee

Andrews, William F.....Memphis
Crane, Paul S.....Nashville
Crutcher, Richard R.....Nashville
Curtis, Lynn F.....Knoxville
Downie, Kenneth E.....Memphis
Dyer, David P.....Ridgetop

Tennessee—Continued

Garrett, Sam Y.....Dixon Springs
Hall, James B.....Nashville
Hutcherson, William P.....Cookeville
(name changed from Koehn)
McFarland, T. C. Jr.....Fountain City
Miller, Fox.....Memphis
Morison, James H. S.....Mountain Home
Truebger, Harold M.....Nashville
Wheeler, Joseph E.....Memphis
Woods, John R.....Nashville

Texas

Adamo, Dominick C.....Houston
Alston, Herbert M.....Mercedes
Battle, William D.....Dallas
Betts, Floyd G., Jr.....Wharton
Bowden, Andy J., Jr.....Weatherford
Coussons, Charles W.....Hemphill
Daily, Herschel.....Richmond
Danforth, Duncan R.....Texas City
Daniel, Dubose B.....Waskom
Daniell, Charles B.....Brownfield
Davis, James H.....Fort Worth
Dawson, William D.....Mathis
Estrada, Ramiro P.....San Antonio
Faubion, Darrell B.....Dallas
Graham, James M.....Sweetwater
Granger, Wayne H.....Dallas
Granville, George E.....Brady
Hafer, William F. L.....Clifton
Haley, Arvel E.....Dallas
Hallmark, James A.....Fort Worth
Hill, Lucius D. III.....San Antonio
Jones, Edgar F. Jr.....Aransas Pass
Klingensmith, W. R., Jr.....Amarillo
Longino, Joseph B.....Sulphur Springs
McKemie, Jack F.....Bryan
McLaurin, Robert L.....Dallas
Meyer, Walter B.....Hondo
Meynier, Maurice J. Jr.....Houston
Murphy, Maurice.....Marshall
Nemir, Paul Jr.....Navasota
Olin, Walter G. Jr.....Houston
Page, Joseph H.....Houston
Perkins, Robert B.....Dallas
Rice, Lee R.....Marshall
Robison, Edward W.....Austin
Rohrer, Vern.....Springtown
Ross, Raleigh R.....Lockhart
Rothenberg, Lionel H.....Wharton
Sibley, D. Jacobi Jr.....Fort Stockton
Smith, Jack C.....Dallas
Swanson, John D. Sr.....El Paso
Taylor, Surse J. Jr.....McKinney
Velinsky, Morris.....Kilgore
Walker, James N.....Munday
Watman, Morris.....Waco
Wiesner, Jerome J.....Rowena
Wright, Thomas R.....El Paso

Utah

Deck, Melvin D.....Provo
Hall, Eugene Y.....Salt Lake City
Learned, Leland O.....Salt Lake City
Petersen, Ralph C.....Hyrum
Pond, Vaughn M.....Logan
Seavers, Ray Jr.....Salt Lake City

Vermont

Beck, Gerhard T.....Burlington
Brooks, George W.....Burlington
Corley, John P.....Burlington
French, Paul K.....Burlington
Howard, Ethan V. Jr.....Burlington
Johnston, William H.....St. Johnsbury
Keenan, Thomas A.....Rutland
Moore, John F.....Fair Haven
Moskovitz, Harold.....Burlington
Pratt, George O.....Richford
Prior, Jack T.....St. Albans
Robinson, Lindsay E.....Brattleboro

Virginia

Bray, Maurice M.....Charlottesville
Brecher, George.....Arlington
Davis, James L.....Waynesboro
Davis, Thomas N. III.....Lynchburg
Dillard, George H. L.....Richmond
Farragut, Loyall D.....Alexandria
Fitzgerald, Walter C.....Crewe
Frostick, Herbert W.....Richmond
Laibstain, Alter.....Norfolk
Laibstain, Herman.....Norfolk
McCall, Fred C.....Norton
Rabil, Pierre J.....Roanoke
Shelburne, Robert C.....Christiansburg
Vick, Herbert W.....Portsmouth
Vranian, George.....Richmond
Walker, Clifford L.....Arlington
Young, Charles G.....Roanoke

Washington

Brougham, Milton F.....Seattle
Burt, Robert R.....Tacoma
Buttorff, Douglas P.....Tacoma
Deitrich, Carlisle.....Tacoma
Gunther, Noel M.....Seattle
Hanford, Russell B.....Spokane
Hauser, Max H.....Spokane
Olson, Hilding H.....Bow
Seibly, Walter W.....Clarkston
Sweet, Paul W.....Centralia

West Virginia

Davis, Henry C.....Bluefield
Gray, James H.....Clendenin
Jones, Ralph Jr.....Parkersburg
Kizinski, Alexander B.....Parkersburg
Neely, Richard C. Jr.....Hinton
Weaver, Neill K.....Parkersburg
Wiesenfeld, Benjamin.....Huntington

Wisconsin

Bender, Roger I.....Milwaukee
Buszkiewicz, Ted S.....Milwaukee
Dametz, John H.....Muscoda
Engle, Howard A.....Milwaukee
Foseid, Oscar F.....Neenah
Galler, Fred O.....Milwaukee
Gurjian, Leon K.....Madison
Holman, James H.....Sheboygan
Kurzon, Alvin M.....Milwaukee
LaBissoniere, Paul G.....Milwaukee
Levin, Jack J.....Milwaukee
Litzow, Louis T.....Milwaukee
Lorenz, Albert A.....Viroqua
Niles, Earl W.....Whitewater
Osborne, Rollin R.....Hartford
Peterson, Leo W.....Sun Prairie
Rydell, John R.....Superior
Sachs, Kurt.....Milwaukee
Smith, Gibson C.....Madison
Sverdlin, Abe A.....Milwaukee
Wangeman, Clayton P.....Madison

Wyoming

Black, Norman R.....Cheyenne
Crisman, Homer R.....Lander
Hoadley, Joseph E.....Cheyenne
Vonburg, Vernon R.....Basin

Canal Zone

Byrd, Jessee L.....Cristobal
Ellington, Jesse C.....Ancon
Phillips, Herbert L.....Balboa Heights
Wright, George R.....Ancon

France

Rogers, Neil C.....Paris

Hawaii

Wong, Ah Yet.....Hilo

Puerto Rico

Carrera, Manuel E.....Fajardo
Colon-Yordan, E.....Ponce
Roure, José Angel.....Quebradillas

Medical News

(Physicians will confer a favor by sending for this department items of news of general interest: such as relate to society activities, new hospitals, education and public health. Programs should be received at least two weeks before the date of meeting.)

COLORADO

School for Medical Record Librarians.—The University of Colorado, Boulder, will inaugurate September 1 an additional school for medical librarians which will lead to a Bachelor of Science degree from the school of business. Students will spend the first three years in the university proper and the fourth year of twelve months in the medical record library of Colorado General Hospital, Denver. The plans were drawn with the advice of the Educational Board of the American Association of Medical Record Librarians.

CONNECTICUT

Dr. Winternitz Acting Director of Research Fund.—The Jane Coffin Childs Memorial Fund for Medical Research has announced the appointment of Dr. Milton C. Winternitz, New Haven, Conn., as acting director of the Board of Scientific Advisers to succeed Dr. Stanhope Bayne-Jones, who has accepted appointment as president of the joint administrative board of the New York Hospital-Cornell Medical Center (THE JOURNAL, June 7, p. 547). Dr. Bayne-Jones, director of the fund since its establishment June 11, 1937, will continue as a member of the Board of Scientific Advisers. The tenth anniversary of the fund was observed June 13 at a dinner in New Haven, Conn., attended by representatives of the U. S. Public Health Service, National Cancer Institute, Donner Foundation, Anna Fuller Fund, American Cancer Society, Committee on Growth of the National Research Council, Connecticut State Cancer Society and Yale University. The fund was established by gifts of $3,300,000 in trust to Yale University by the late Starling W. Childs and Miss Alice S. Coffin, New York. Dr. Bayne-Jones was the principal speaker. "The fund," he said, "has regarded the cancer problems as problems of growth and has supported work in fundamental fields of chemistry, biology, viruses, pathology, genetics and medicine." Of the eighty-one cancer projects it has supported, 78 per cent of those now in operation have been supported for three years or longer and some as long as ten years with provision up to twelve years. A fellowship program was put into operation in 1944 and twelve research fellowships have been granted. The fund is not restricted to any institution, although 40 to 60 per cent of its annual appropriations have been given to Yale. The present ratio is about evenly divided between cancer research at Yale and at other institutions in this country and abroad.

DISTRICT OF COLUMBIA

Medical Research at George Washington University.—George Washington University School of Medicine is operating this year the largest research program in its history under grants totaling $150,000. The largest grant, $40,000, is for research in anesthesia and the use of penicillin and streptomycin. Projects are under way for the study of drugs to treat typhus fever, Rocky Mountain spotted fever, rheumatic fever, tumors, arthritis and for relief of muscle spasms. Other projects deal with the study of hypertension, vitamins C and E, protein metabolism and therapy, cardiovascular disease and vascular surgery.

ILLINOIS

Federal Grant for Research in Psychology.—The University of Illinois, Urbana, has been given grants from the federal government totaling $87,850 for research and training in the field of psychology. The funds include $4,000 from the U. S. Public Health Service for fellowships and $7,250 for additional staff.

Fifty Years in Practice.—Dr. Robert W. Binney, Granite City, was given a dinner in honor of his completion of fifty years in the practice of medicine by the Tri-Cities Medical Society. Society members, classmates of Dr. Binney, representatives of industrial plants and the Chamber of Commerce of Granite City attended. The veteran doctor was presented a bronze plaque commemorating the occasion.

Chicago

Personal.—Dr. Fred L. Glenn was cited by the Kiwanis Club of Austin May 29 for unselfish service to various civic groups with which he has been actively associated.——Dr. William D. McNally, coroner's toxicologist for twenty-one years, resigned June 27.

Cook County Interns to Be Country Doctors.—A class of 58, including 4 women, began a two year internship at Cook County Hospital July 1 designed to train them as general practitioners. Dr. Karl A. Meyer, medical superintendent of the hospital, said that 99 per cent of the class when asked where they intended to practice said they would go into small communities where the need is greatest. This is the first class to enter the hospital under the new required two year internship regulation (THE JOURNAL, March 15, p. 791).

Research in Nucleonics.—Construction at the southwest corner of Ellis Avenue and 56th Street of a $1,250,000 ion accelerator building, first unit in the University of Chicago's $10,000,000 program for basic research in nucleonics, began July 9. This laboratory will house a 100 million volt betatron and two cyclotrons of the most modern construction. The first building in the proposed housing program for the Institute for Nuclear Studies, the Institute of Radiobiology and Biophysics and the Institute for the Study of Metals will be a one story structure with basement. The facilities for medical research will be used by the Institute of Radiobiology and Biophysics and the division of roentgenology of the department of medicine. One problem in prospect is a study of the effects of positive ion beams on deep-seated cancer. The accelerator building, to be ready for occupancy next spring, will make possible the resumption of advanced basic research, which has been at a standstill since the war. Among the many scientists at the university who will work in the laboratory are Samuel K. Allison, who participated in the first atomic bomb explosion in New Mexico; Enrico Fermi, Nobel prize winner, who was the first to achieve a controlled nuclear chain reaction; Harold C. Urey, discoverer of "heavy water" and Nobel prize winner; Arthur J. Dempster, discoverer of U-235; Edward Teller and Willard F. Libby, who with associates, according to the university's press release, "lately discovered radiocarbon in all living matter"; Raymond E. Zirkle, Dr. Austin M. Brues, Kenneth S. Cole and Konrad E. Bloch. Dr. Paul C. Hodges, roentgenologist, will have charge of the medical aspects of the program. The ion accelerator building will be of fireproof construction. The large cyclotron will be placed in the pit, so that its beam will be underground and not detectable by the betatron, which, together with the present cyclotron, will be enclosed in concrete walls. Five laboratories, offices for the medical staff and examining rooms for patients will be located the full length of the building. Offices, laboratories, conference room and drafting room for the Institute for Nuclear Studies and an office and laboratory for the Institute of Radiobiology and Biophysics will be located on the east balcony.

INDIANA

Appoint Assistant to Dean.—John J. Mahoney, Ph.D., formerly on the University of Illinois faculty, has been appointed assistant to the dean and assistant professor of experimental medicine at the Indiana University Medical Center, Indianapolis. Dr. Mahoney received his Ph.D. degree from the State University of Iowa, Iowa City. He has been an instructor in zoology and research associate in zoology at the State University of Iowa, Iowa City.

KANSAS

Changes in Health Officers.—The state board of health has announced the following changes in health officers: Dr. Samuel B. Muller, Pittsburg, has been appointed city health officer to replace Dr. Otto B. Kiehl. Dr. John M. Porter, Concordia, resigned as health officer of Cloud County after his appointment as a member of the Kansas State Board of Health; Dr. Frank Kinnamon, Concordia, will succeed him. Dr. Robert M. Heilman, Topeka, formerly director of the industrial hygiene division of the state health department, has returned to the Kansas State Board of Health as director of the newly created division of hospital survey and construction.

KENTUCKY

Division of Psychosomatic Medicine.—Grants totaling $49,304 for three departments of the University of Louisville School of Medicine have been received from the U. S. Public Health Service to provide funds for research and expansion of present facilities. A division of psychosomatic medicine in the

school is to be organized, and two fellowships of $3,600 in that field will be offered. The grant will make possible the addition of one associate professor with special training, one society worker, one psychologist and two part time specialists. The department of anatomy received $9,960 for a study of toxemia of pregnancy in guinea pigs. In the department of pharmacology a $7,344 grant will be used to study the peptic ulcer problem.

MAINE

State Medical Election.—At the annual meeting of the Maine Medical Association at York Harbor, June 22-24, Dr. Stephen A. Cobb, Sanford, was elected president; Dr. Forrest B. Ames, Bangor, president-elect, and Dr. Frederick R. Carter, Portland, secretary-treasurer.

Maine Psychiatric Society.—This society was founded at the Augusta State Hospital, June 19, for group consideration of all matters relating to psychiatry in the state. Officers elected were Dr. Carl J. Hedin, Bangor, medical superintendent, Bangor State Hospital, president; Dr. Frederick R. Carter, Portland, vice president; Dr. Francis H. Sleeper, medical director of the Augusta State Hospital, Augusta, secretary-treasurer.

MARYLAND

Gift to Johns Hopkins University.—Johns Hopkins University, Baltimore, announced on July 11 a gift of $1,000,000 from Undersecretary of State and Mrs. Will L. Clayton for medical research. The terms of the gift permit it to be used for any research regarded as most important to the department of medicine "in the present state of development of the medical sciences."

Adolf Meyer Laboratory of Neurology.—The medical advisory board of the Seton Institute and the Sisters of Charity of St. Vincent de Paul, Baltimore, have announced the gift from Dr. Adolf Meyer, Baltimore, of his entire personal collection of neuroanatomic and neuropathologic material, consisting of some sixty large boxes of serial sections from crucial human case material as well as material of a comparative nature. Included in the gift is an exhaustive card index file of neurologic subjects. The institute plans to use this material as the nucleus about which to develop a laboratory, which will be called the Adolf Meyer Laboratory of Neurology, for the study of neuroanatomy, neurophysiology and neuropathology. The material will soon be in available form for the instruction of candidates for the American board examinations in neurology. Seton Institute, the former Mount Hope Retreat, provides facilities for the training of psychiatric interns and for three years of postgraduate service required of the specialists certified by the American Board of Psychiatry and Neurology. Dr. Wendell S. Muncie, Baltimore, is chairman of the medical advisory board.

MASSACHUSETTS

Personal.—Dr. Frank H. Lahey, founder and director of the Lahey Clinic, Boston, received the honorary degree of Doctor of Science June 18 from Northwestern University, Evanston, Ill.

Lectures in Public Health.—"What's New in Public Health," a series of twelve lectures for public health workers in the North Metropolitan District of the Massachusetts Department of Public Health, has just been completed. Dr. A. Daniel Rubenstein, health officer of the district, planned the lectures to bring professional workers up to date in this field. The expenses of the course were assumed by the Middlesex Health Association, and the Harvard School of Public Health offered its facilities. Members of the Harvard School of Public Health faculty, the staff of the Massachusetts Department of Public Health and national public health experts gave the lectures to sixty-seven public health workers including board of health, school and industrial nurses, board of health agents and health educators.

Dr. Eaton Appointed Associate Professor.—Dr. Monroe D. Eaton, director of research laboratory of the state department of public health at the University of California, Berkeley, was appointed associate professor of bacteriology and immunology in Harvard Medical School, effective July 1. A graduate of Harvard Medical School, Boston, 1930, he served for three years as assistant in bacteriology and immunology in that school. From 1933 to 1936 he was an instructor in bacteriology at the Yale University School of Medicine, New Haven, Conn., and the following year was assistant professor of bacteriology and immunology in Washington University School of Medicine, St. Louis. Since 1937 he has been a member of the staff of the International Health Division of the Rockefeller Foundation and director of the research laboratory for the state of California.

MICHIGAN

Dr. Stapleton Retires.—Dr. William J. Stapleton Jr., Detroit, actively connected with teaching at the Detroit College of Medicine from 1907 and since 1914 professor of medical jurisprudence, as well as assistant, associate or acting dean since 1935, has retired. In addition to his teaching he has been engaged in active practice of medicine for forty-seven years. He received his M.D. from Michigan College of Medicine and Surgery, Detroit, in 1900, his Ph.G. from the department of pharmacy in the Detroit College of Medicine, 1902, and his LL.B. from Detroit College of Law, 1907.

Whooping Cough in Michigan.—According to Dr. William DeKleine, commissioner, Michigan Department of Health, Lansing, whooping cough, which is now appearing in the state at the rate of 200 cases a week, has taken the lives of 33 children during the first five months of this year. Eighteen of these deaths were of babies under 6 months of age.. A total of 5,312 cases have been reported in the state since January 1. The Michigan Department of Health recommends that all babies at 6 months of age be given four weekly doses of vaccine, which is made by the Michigan Department of Health laboratories and distributed without charge to physicians in the state.

Course in Food Handling.—The School of Public Health, University of Michigan, gave a course on food handling from June 19 to 21 designed to serve the needs of public health workers confronted with problems of food handler education and food establishment sanitation. The course was planned with the assistance of various industries such as drug stores, restaurants and hotels, and the university, the U. S. Public Health Service, Illinois state health department and the National Sanitation Foundation, the chairman of the group being Henry F. Vaughan, Dr.P.H., dean of the School of Public Health, University of Michigan. The course was attended by health workers from various parts of the United States, and three day discussions were presided over by Dr. Vaughan, Dr. Herman N. Bundesen, Chicago Board of Health, and Jerome Trichter, director of the Bureau of Food and Drugs, New York.

Rheumatic Fever Program.—Rheumatic fever, which kills five times as many children as infantile paralysis and cripples many more, is being attacked on a statewide basis in Michigan under a program set up by the committee from the state medical association. Joint sponsors of the program are the Michigan State Medical Society, Michigan Crippled Children Commission and the Michigan Society for Crippled Children and Disabled Adults, Inc. The objectives are (1) recognition and proper management of every case in Michigan, (2) determination of the incidence of the disease, (3) provision for special hospital and convalescent care centers, (4) education and continuous recourse to recent advances in research and (5) lay education and lay group cooperation. Nine rheumatic fever consultants and diagnostic centers have been established in the state and additional centers are to be set up in Muskegon, Wayne County, Battle Creek, Saginaw and Port Huron. Where the families cannot afford it, the small laboratory cost fee is paid through the Michigan Crippled Children's Commission. New cases are reported to the state health department, and monthly reports are submitted to the central office of the Michigan State Medical Society by the centers. Physicians return cases to the centers at intervals for reappraisal.

MINNESOTA

Research Professor in Rheumatic Fever.—Plans have been formulated to establish at the University of Minnesota, Minneapolis, a professorship which will direct research in heart diseases and rheumatic fever of children. The American Legion of the state of Minnesota is embarking on a campaign to secure $500,000 from its members to maintain this chair as a memorial to veterans of World Wars I and II.

Townspeople Fete Dr. Grinnell.—Hundreds of townspeople as well as patients throughout the surrounding country attended a homecoming in Preston, Minn., recently in honor of Dr. Wendell B. Grinnell, who has practiced medicine for forty-seven years. Dr. Grinnell began practicing in Fillmore County in 1900 and one year later moved to Preston. For many years he owned and operated his own hospital.

MONTANA

New Cancer Service.—The Cancer Committee of the Montana State Medical Association is inaugurating a program to provide medical service and hospital expenses for medically indigent cancer patients, interest free loans for people temporarily unable to pay, tissue examinations and outpatient care. The project has been worked out in cooperation with the state

board of health and the department of public welfare, which has volunteered to do the case work. Dr. Eugene Hildebrand, Great Falls, is chairman of the state division's executive committee, which will supervise the operation of the project.

NEW YORK

Dr. Hilleboe Appointed State Health Commissioner.—Governor Dewey appointed Dr. Herman E. Hilleboe, Assistant Surgeon General of the U. S. Public Health Service, to take over the duties of the New York State Health Commissioner on July 1, succeeding Dr. Edward S. Godfrey, who has retired (THE JOURNAL, May 31, p. 472). Dr. Hilleboe graduated from the University of Minnesota Medical School in 1931 and received a Master of Public Health degree in 1935 at Johns Hopkins School of Hygiene and Public Health, Baltimore. For a time he engaged in general practice in Swanville, Minn., and since 1935 has spent full time in public health work specializing in tuberculosis control. In 1939 he was appointed senior surgeon in the regular corps of the Public Health Service. From 1939 to 1942 he was chief of the medical unit, Minnesota Division of Social Welfare, in St. Paul, on loan from the Public Health Service, and has since been in tuberculosis control work in the Public Health Service, Washington, D. C. He has been Assistant Surgeon General since Nov. 1, 1946. Dr. Hilleboe is a member of the board of directors of the National Tuberculosis Association, a member of the Council of the American Trudeau Society and of the Board of Regents of the American College of Chest Physicians and representative on the Council of the International Union Against Tuberculosis. He is professorial lecturer in tuberculosis at the George Washington University Medical School, Washington, D. C., and adjunct professor of medicine, Georgetown University, Washington. He is the American member of the Expert Committee on Tuberculosis of the World Health Organization Interim Commission.

New York City

Cancer Professorship.—Cornell University Medical College has established a professorship of cancer with an annual endowment of $15,000 for five years. The sum represents the first allocation of funds from the 1947 campaign of the New York City Committee of the American Cancer Society.

Venereal Disease Seminar for Practicing Physicians.—A new series of lectures for practicing physicians on the diagnosis, treatment and management of venereal diseases will start at the New York City Department of Health, 125 Worth Street, Manhattan, on Saturday morning, September 13. Meetings will be held every Saturday morning at 10:30 through December 20. Experts in special fields of venereal disease control will participate in the seminar. No registration or fee for the course will be required. Physicians who attend may be reached by their offices at WOrth 2-6900, extension 460.

Rehabilitation Wards Opened.—Mayor William O'Dwyer formally opened two new wards at New York's municipal Bellevue Hospital July 7, designed to provide a complete program of rehabilitation for disabled civilians similar to that available to disabled veterans through the Veterans Administration. The two wards, which will accommodate 80 patients, are claimed to comprise the first rehabilitation program in any civilian general hospital in the nation. The new program is an outgrowth of a grant of $250,000 in 1944 by the Baruch Committee on Physical Medicine to the New York University College of Medicine, which is responsible for the professional services of the new department. Dr. Howard A. Rusk, head of the department of rehabilitation and physical medicine, New York University College of Medicine, was appointed consultant in rehabilitation to the department of hospitals of the city health department. He will supervise the expansion of the program into other city hospitals. It is expected that the 80 rehabilitation beds in Bellevue will eventually be expanded to about 600 when the new Bellevue Hospital is constructed.

NORTH CAROLINA

University Promotions.—Duke University School of Medicine, Durham, has announced the promotion of Dr. Lenox D. Baker to professor of orthopedics; Dr. Jasper L. Callaway to professor of dermatology and syphilology; Dr. Robert W. Graves to professor of neurology; Dr. M. Barnes Woodhall to professor of neurosurgery; Philip Handler, Ph.D., to associate professor of biochemistry, and Dr. Jerome S. Harris to associate professor of pediatrics.

Appoint College Physician.—Dr. Cary F. Irons, Greenville, has been appointed physician at East Carolina Teachers College, Greenville, effective September 1, replacing Dr. Frederick P. Brooks, who has held the position since 1935. Dr. Irons received his M.D. degree at the Medical College of Virginia, Richmond. During World War II he served in the medical reserve corps and was discharged as a major. Dr. Brooks will devote full time to private practice.

SOUTH CAROLINA

Health Officer Assigned.—Dr. Carolyn H. Callison, formerly health officer for Greenwood and McCormick counties, has returned to the state board of health after receiving her Master of Public Health degree from Columbia University College of Physicians and Surgeons, New York, and has been assigned to the Charleston County Health Department. She also served four years as a county health officer in Alabama.

TEXAS

Appointed to State Board.—The appointment of the following doctors to the state board of medical examiners has been announced: Drs. McKinley H. Crabb, Forth Worth; Herschel F. Connally, Waco, and Neil D. Buie, Marlin, were reappointed. Dr. Ralph H. Peterson, Wichita Falls, will serve his first term.

New Surgical Unit.—A special surgical unit for plastic and maxillofacial surgery and neurosurgery is being developed at the University of Texas Medical Branch, Galveston, to be opened in September. This unit was planned by the late Dr. Albert O. Singleton and includes an operating suite with x-ray appliances, offices, wards and private rooms. The new unit will be under the direction of Dr. Truman G. Blocker Jr., professor of plastic and maxillofacial surgery, and Dr. Samuel R. Snodgrass, associate professor of neurosurgery.

Drs. Blattner and Brown to Head Departments.—Baylor University College of Medicine, Dallas, has announced the appointment of Dr. Russell J. Blattner, associate professor of pediatrics, Washington University School of Medicine, St. Louis, as professor and chairman of the department of pediatrics, beginning September 1. Dr. Blattner received his M.D. degree from Washington University School of Medicine, St. Louis, in 1933. He was an intern in 1934, an assistant resident in 1935 and a resident, all at St. Louis Children's Hospital. Dr. Warren T. Brown, formerly associate professor of psychiatry at Yale University School of Medicine, New Haven, on July 1 was made professor of psychiatry and chairman of the department of neuropsychiatry at Baylor University. He received his medical degree from the University of Texas Medical Branch, Galveston, in 1933 and went to Yale University School of Medicine as assistant professor of psychology and mental hygiene.

UTAH

University Appointments.—The University of Utah School of Medicine, Salt Lake City, has announced the following additions to its medical staff: Thomas F. Dougherty, Ph.D., has been appointed professor in the department of anatomy (histology). Dr. William Moretz, recently instructor in surgery at Rochester University, Rochester, N. Y., has joined the staff as assistant professor in the department of surgery, succeeding Dr. Randolph T. Shields, who has left for China. Dr. Hans Reich of the University of Basel, Basel, Switzerland, will join the staff to assist in a cancer research project now under way in the department of biochemistry, financed by grants from the Cancer Research Division of the Donner Foundation and the U. S. Public Health Service.

WASHINGTON

Reinstate Course for Medical Speakers.—King County Medical Speakers Club will present a special course consisting of ten consecutive Monday night sessions beginning September 15 and a similar series beginning Wednesday, September 17. The special course will feature speech fundamentals, how to write a speech, manuscript reading, radio technic and parliamentary law. The course, under Mr. Clayton L. Walton, state parliamentarian for the Junior Chamber of Commerce, was presented in 1941 and 1942 also. Further information may be obtained by writing the King County Medical Society, 118 Cobb Building, Seattle 1.

GENERAL

Special Society Elections.—At the annual meeting of the American Association on Mental Deficiency in St. Paul, May 27-31, Lloyd Yepson, Ph.D., Trenton, N. J., was elected president; Dr. Edward J. Humphreys, Columbus, Ohio, president-elect and editor of the journal; Dr. Neil A. Dayton, Mansfield Depot, Conn., secretary-treasurer. Final plans are outlined for the first International Congress on Mental Deficiency, which will be held in Boston at the Hotel Statler May 11-15, 1948.—At the annual meeting of the American College of Radiology in

Atlantic City June 8 Drs. Edwin C. Ernst, St. Louis, was elected president, Charles L. Martin, Dallas, Texas, vice president, Warren W. Furey, Chicago, treasurer and Mac F. Cahal, Chicago, executive secretary.

Gastroscopic Club Changes Name.—At the recent meeting of the American Gastroscopic Club in Atlantic City the name of the organization was changed to the American Gastroscopic Society. The group elected Drs. Herman J. Moersch, Rochester, Minn., president; James L. Borland, Jacksonville, Fla., vice president, and Joseph B. Kirsner, Chicago, secretary-treasurer.

Research in Multiple Sclerosis.—Contribution of $25,000, made by Mrs. Percy S. Straus, New York, has been announced by the Association for Advancement of Research on Multiple Sclerosis, Inc. With this contribution the initial goal of $100,000 for research on multiple sclerosis has been met. The first grant made by the association was for $64,350 for work in the field of allergy (THE JOURNAL, June 21, p. 722).

National League of Nursing Education.—The National League of Nursing Education will hold its fifty-first convention in Seattle September 8-11. The theme of the convention will be "Nursing Education for Public Service." The Hawaii League of Nursing Education will be formally accepted into the National League at this time. Details may be obtained from the National League of Nursing Education, 1790 Broadway, New York 19.

Dr. Kato Awarded Prize for Painting.—Dr. Hideo H. Kato, intern at the General Hospital, Louisville, Ky., won the $250 prize of the American Physicians Art Association at the exhibit held in Atlantic City in June. The painting dedicated to "combat medics" is entitled "Beyond the Call of Duty." Dr. Kato, a young Japanese American, graduate of the University of Louisville School of Medicine in 1941, served in the U. S. Army overseas twenty months. He had no previous training in art and began painting as a hobby after the war.

Publicity Campaign Against Tuberculosis.—The National Tuberculosis Association reports that the Advertising Council, Inc., will launch a nationwide publicity campaign urging periodic chest x-rays for all adults. The campaign, approved by the National Tuberculosis Association in March, will use newspapers, magazines, radio and display to drive home the necessity of early diagnosis. Space and time will be donated through the council by sponsors and advertisers who are represented in the council and whose contributions mainly support it.

The Banting Lecture.—Dr. George H. A. Clowes, Ph.D., director emeritus of the Lilly Research Laboratories, delivered the annual Banting memorial address of the American Diabetes Association at its recent meeting in Atlantic City and was awarded the Banting Medal, given in recognition of distinguished service in the field of diabetes. Under Dr. Clowe's direction the laboratories cooperated with the University of Toronto Faculty of Medicine, Ontario, and Drs. Banting and Best in the early development of insulin of sufficient purity and stability to permit its widespread clinical use throughout the world.

Scientific Attack on Problem Drinking.—The first large scale nationwide attack on problem drinking will be launched this fall by the Research Council on Problems of Alcohol. Organized ten years ago, the Research Council has been sponsoring individual research projects among scientific institutions to get at the fundamental causes of problem drinking, which affects the lives of over 750,000 persons in the United States. The primary objectives of the council are not only to learn more about these fundamental causes but to develop effective methods of treatment and prevention. It hopes to establish research diagnostic and treatment centers in leading medical schools and their affiliated hospitals. One has already been established at Cornell University Medical College–New York Hospital under a grant of $150,000 covering a five year period.

Radio Program on Rural Medical Service.—The Allis-Chalmers Manufacturing Company, West Allis, Wis., sponsors of the National Farm and Home Hour broadcast each Saturday noon over the radio facilities of the National Broadcasting Company and its independent affiliated stations, will deal with the problem of rural medical service in a special broadcast scheduled for August 2 at 12 noon, Central Standard time. Special guests on this program, representing the rural physician and his wife, will be Dr. and Mrs. Charles C. Collester of Spencer, Iowa. Arrangements for Dr. and Mrs. Collester to appear on the program were made by the Bureau of Health Education of the American Medical Association through the Iowa State Medical Society. Dr. and Mrs. Collester will travel to Chicago to broadcast from WMAQ as the special guests of the sponsors.

International Cancer Research Congress.—The fourth International Cancer Research Congress will be held in St. Louis, September 2-7, with headquarters at the Jefferson Hotel, under the sponsorship of the Union Internationale Contre le Cancer and the American Association for Cancer Research. General sessions will include presentations on the following topics:

September 3. General Aspects of Cancer Research; Cancer Surgery; Radiation Therapy of Cancer; Etiology of Cancer.
September 4. Etiology of Cancer (Carcinogens); Chemistry in Relation to Cancer.
September 5. Hormones and Cancer; Biology of Cancer.
September 6-7. Nuclear Physics in Relation to Cancer; Cancer and the Host.

Edmund V. Cowdry, Ph.D., Barnard Free Skin and Cancer Hospital, St. Louis, is president of the Congress, Dr. Shields Warren, New England Deaconess Hospital, Boston, is chairman of the Program Committee, and Dr. Axel N. Arneson, Mellinckrodt Institute of Radiology, St. Louis, is chairman of the Committee on Local Arrangements.

Prevalence of Poliomyelitis.—Reports of cases of poliomyelitis for the periods indicated have been received from the division of public health methods, U. S. Public Health Service:

Division and State	Week Ended July 12, 1947	Week Ended July 13, 1946	Median, 1942-1946	Total to July 12, 1947	Total to July 13, 1946	Total * March 16-July 12, 1947	Total * March 17-July 13, 1946
New England:							
Maine	0	0	1	7	3	4	3
New Hampshire	0	4	0	5	7	1	6
Vermont	0	0	0	8	7	0	3
Massachusetts	3	0	2	15	7	8	3
Rhode Island	1	0	0	7	0	6	0
Connecticut	1	4	3	9	19	6	11
Middle Atlantic:							
New York	8	15	15	97	128	61	95
New Jersey	2	6	2	22	27	16	21
Pennsylvania	7	1	3	28	33	14	21
East North Central:							
Ohio	5	13	10	33	60	23	45
Indiana	2	3	3	23	21	7	16
Illinois	9	23	6	58	102	36	94
Michigan	2	11	4	40	27	16	19
Wisconsin	0	4	1	24	20	0	7
West North Central:							
Minnesota	5	40	1	30	81	17	80
Iowa	2	8	2	20	43	12	34
Missouri	5	14	4	29	49	19	42
North Dakota	2	1	0	20	4	15	4
South Dakota	0	5	0	2	8	0	8
Nebraska	5	12	0	35	19	28	18
Kansas	0	18	5	20	55	2	51
South Atlantic:							
Delaware	0	0	0	1	1	1	1
Maryland	0	3	1	12	8	6	6
District of Columbia	0	1	1	3	2	1	1
Virginia	4	3	3	23	13	13	7
West Virginia	1	1	1	6	9	2	6
North Carolina	4	1	1	26	38	11	31
South Carolina	2	3	3	6	14	5	11
Georgia	6	2	2	24	48	17	40
Florida	3	24	2	51	338	26	289
East South Central:							
Kentucky	2	5	5	15	29	14	26
Tennessee	0	1	5	18	23	6	14
Alabama	0	14	6	23	136	10	133
Mississippi	0	6	2	22	51	5	34
West South Central:							
Arkansas	2	20	7	22	58	12	46
Louisiana	1	18	1	34	104	16	85
Oklahoma	1	15	8	21	64	10	58
Texas	6	54	45	78	391	53	363
Mountain:							
Montana	0	6	0	4	30	2	14
Idaho	2	0	0	15	1	5	1
Wyoming	2	0	0	6	2	3	1
Colorado	2	31	1	18	135	13	134
New Mexico	0	1	1	6	17	3	16
Arizona	1	1	0	12	11	9	8
Utah	1	0	0	6	13	1	7
Nevada	0	0	0	0	0	0	0
Pacific:							
Washington	3	5	2	28	63	20	30
Oregon	1	5	2	10	10	5	6
California	22	25	22	399	261	250	174
Total	125	427	...	1,421	2,590†	810	2,123
Median, 1942-1946	...	...	297	1,679			

* Last two columns show reported incidence since approximate seasonal low week (week ended between March 15 and 21).
† Figures changed by corrected reports.

Deaths

Linda Bartels Lange, Philadelphia; Johns Hopkins University School of Medicine, Baltimore, 1911; professor of bacteriology and immunology at the Woman's Medical College of Pennsylvania from 1937 to 1940; fellow in pathology and bacteriology at the Rockefeller Institute for Medical Research, New York, from 1912 to 1914 and pathologist and director of the laboratory at the Howard A. Kelly Hospital, Baltimore, 1914-1915; instructor in pathology at the University of Wisconsin Medical School in Madison, 1915-1916, and instructor and assistant in medicine at the Johns Hopkins University School of Medicine in Baltimore from 1916 to 1919; fellow, instructor and associate in bacteriology at the Johns Hopkins University School of Hygiene and Public Health in Baltimore from 1919 to 1927, when she became associate professor, serving until 1937; died April 24, aged 65, of pneumonia.

Delbert Oscar Kearby, Indianapolis; born in Rivervale, Ind., Dec. 31, 1878; Indiana Medical College, School of Medicine of Purdue University, Indianapolis, 1906; formerly professor of bronchoscopy and esophagoscopy at the Indiana University School of Medicine, where he had been in charge of the department; at one time served in the house of representatives of the state of Washington; specialist certified by the American Board of Otolaryngology; fellow of the American College of Surgeons; past president of the Indiana Academy of Ophthalmology and Otolaryngology; served on the staffs of the Indianapolis City Hospital, Robert W. Long Hospital, St. Vincent's Hospital, James Whitcomb Riley Hospital for Children and the Methodist Hospital, where he died April 10, aged 68, of rupture of the abdominal aorta.

George Edward Perkins, Boston; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1913; member of the American Medical Association; served during World War I; during World War II served as a lieutenant colonel in the medical corps, Army of the United States, directing the venereal disease control program of the Sixth Army Corps and a consultant with rating boards under the Veterans Administration; acting director, division of venereal diseases, Massachusetts Department of Public Health; consultant at the Boston Dispensary and Massachusetts Memorial Hospitals; died in the Burbank Hospital, Fitchburg, April 3, aged 68, of injuries received in an automobile accident.

Joseph Johns ✦ Ionia, Mich.; born on May 10, 1882; American Medical Missionary College, Battle Creek, Mich., and Chicago, 1910; past president of the Ionia-Montcalm Counties Medical Society; an examiner for the draft board of Ionia County during World War II; on the staffs of the Michigan Reformatory and the Ionia State Hospital; member and past president of the staff of the Ionia County Memorial Hospital; member of the staff of the Clinton Memorial Hospital, St. Johns; visiting surgeon to St. Mary's Hospital and the Blodgett Memorial Hospital in Grand Rapids, where he died April 6, aged 64, of cerebral hemorrhage.

John Daniel Donovan, Detroit; St. Louis University School of Medicine, 1935; interned at the Providence Hospital; member of the American Medical Association, American Association of Industrial Physicians and Surgeons and the Michigan Association of Industrial Physicians and Surgeons; industrial physician for the Ford Motor Company, serving as a member of the Rouge plant industrial hospital staff and during World War II assistant physician in charge of the industrial hospital at the Willow Run bomber plant; died in the Henry Ford Hospital April 18, aged 35, of uremia secondary to sulfadiazine anuria.

John T. Harrington, Waco, Texas; Louisville (Ky.) Medical College, 1887; for many years chairman of the board of trustees and official physician at the Baylor University, where in 1945 the dormitory was named for him; served as president of the board of health of El Paso, as city physician of Waco, as supervisor of Texas penitentiaries and as director of the Epileptic Colony in Abilene; one of the founders of the Hardin-Simmons University at Abilene; on the staffs of the Providence and Hillcrest Memorial hospitals; died April 29, aged 89, of carcinoma of the prostate.

Horace Woodward Carle ✦ St. Joseph, Mo.; Bennett Medical College, Chicago, 1914; past president of the Buchanan County Medical Society; past president and for many years a member of the school board; member of the American Heart Association and the Southern Medical Society; fellow of the American College of Physicians; formerly member and for one year president of the state board of health; affiliated with the Missouri Methodist Hospital and St. Joseph's Hospital; died April 27, aged 57, of pulmonary embolism, chronic heart disease and pneumonia.

Joseph Vincent S. Dauksys, Beacon, N. Y.; Rush Medical College, Chicago, 1926; member of the American Medical Association, Radiological Society of North America and the American College of Radiology; at one time secretary-treasurer of the Mercer County (Ill.) Medical Society; for many years affiliated with Veterans Administration hospitals at various places; major in the medical corps, Army of the United States, during World War II assigned to the Veterans Administration Hospital in Castle Point; died April 6, aged 47, of pulmonary tuberculosis.

William Dietrich Aufderheide ✦ St. Louis; Marion-Sims College of Medicine, St. Louis, 1898; also a graduate in pharmacy; on the staffs of the Alexian Brothers Hospital, Deaconess Hospital and the Lutheran Hospital, where he died April 16, aged 71, of cerebral hemorrhage and hypertension.

Joseph E. Benton, Central City, Neb.; Omaha Medical College, 1897; member of the American Medical Association; on the staffs of St. Francis Hospital in Grand Island and the Hord Memorial Hospital; surgeon for the Union Pacific and Burlington railroads; died in St. Joseph's Hospital in Omaha April 22, aged 71, of carcinoma of the cecum with metastases to the liver.

Thomas Franklin Beveridge ✦ Muscatine, Iowa; Kentucky School of Medicine, Louisville, 1891; affiliated with the Benjamin Hershey Memorial Hospital, where he died April 22, aged 80, of coronary occlusion.

Charles David Bles, New York; Fordham University School of Medicine, New York, 1911; served as medical officer with the 102d Engineers of the New York National Guard, on the Mexican border and during World War I; died in Veterans Administration Hospital number 81, March 5, aged 68.

John A. Boyd ✦ Santa Barbara, Calif.; Western Pennsylvania Medical College, Pittsburgh, 1903; died April 9, aged 69, of coronary occlusion.

John Francis Boyle ✦ Lowell, Mass.; Harvard Medical School, Boston, 1903; member of the New England Obstetrical and Gynecological Society; past president of the Middlesex North Medical Association; on the staff of St. Joseph's Hospital; died April 25, aged 75, of arteriosclerotic heart disease.

Thomas L. Bradley, Warrensburg, Mo.; Washington University School of Medicine, St. Louis, 1896; county jail physician; served as county coroner; died April 10, aged 76, of arteriosclerosis.

Frank Erwin Brinckerhoff ✦ Beloit, Wis.; University of Illinois College of Medicine, Chicago, 1913; fellow of the American College of Surgeons; served during World War I; on the staff of the Beloit Municipal Hospital; died in St. Petersburg, Fla., April 19, aged 56, of coronary occlusion.

Frank Elbert Burbank ✦ Oxford, Iowa; State University of Iowa College of Medicine, Iowa City, 1892; died in the Community Hospital, Pleasantville, April 19, aged 90, of cerebral hemorrhage.

Sidney Raymond Carsley ✦ Palmer, Mass.; University of Vermont College of Medicine, Burlington, 1902; medical examiner for Hampden District; affiliated with the Wing Memorial Hospital in Palmer, Mary Lane Hospital in Ware and the Springfield Hospital in Springfield, where he died April 19, aged 71, of coronary thrombosis.

Levi Bennett Cochran ✦ Hartford, Conn.; University of Pennsylvania Department of Medicine, Philadelphia, 1893; served on the staff of the Hartford Hospital, where he died April 21, aged 79, of coronary sclerosis with infarction.

Morris Coplan ✦ Cleveland; University of Wooster Medical Department, Cleveland, 1897; member of the Cleveland Academy of Medicine; served on the staff of the Mount Sinai Hospital; on the staffs of the Lutheran Hospital and the Fairview Park Hospital, where he died April 3, aged 73, of coronary thrombosis.

Herbert Richard Crase, Los Angeles; Stanford University School of Medicine, San Francisco, 1927; member of the American Medical Association; on the staffs of the California Lutheran Hospital and the Queen of Angels Hospital, where he died February 21, aged 45, of hypertension and uremia.

Ira Alexander Dailey, Micanopy, Fla.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1916; member of the American Medical Association; affiliated with the Alachua County Hospital; died in Gainesville April 14, aged 56, of carcinoma of the bladder.

Lawrence Leroy Davidson ✦ Santa Monica, Calif.; University of Nebraska College of Medicine, Omaha, 1926; on the staffs of the Santa Monica and St. John's hospitals; died in Los Angeles April 21, aged 45, of arteriosclerosis and coronary occlusion.

Samuel H. Dillard, Brookhaven, Ga.; Atlanta Medical College, 1883; died March 16, aged 86, of uremia.

William James Donahue ✠ Newark, N. J.; Long Island College Hospital, Brooklyn, 1909; member of the American Psychiatric Association; served during World War I; received a certificate of appreciation from the War Department for service on the Induction Examining Board in the New Jersey-Delaware Recruiting and Induction District during World War II; on the staffs of the City Hospital, St. Michael's Hospital and Presbyterian Hospital, where he died April 18, aged 63, of carcinoma of the left lung.

Robert P. Donaldson, Pontotoc, Miss.; Memphis (Tenn.) Hospital Medical College, 1901; member of the American Medical Association; for many years county health officer; member of the Selective Service Board during World War II; for many years vice president of the Bank of Pontotoc; died in the Community Hospital, Tupelo, April 30, aged 70, of injuries received in an automobile accident.

Roswell Thomas Eldon, Altoona, Pa.; Jefferson Medical College of Philadelphia, 1891; member of the American Medical Association; served on the staffs of the Mercy and Altoona hospitals; died April 12, aged 79, of coronary thrombosis.

Ord Everman, Indianapolis; Indiana University School of Medicine, Indianapolis, 1908; served during World War I; psychiatrist for the Veterans Bureau and had been stationed at hospitals in various parts of the country; died April 21, aged 71, of coronary occlusion.

Edward Hilts Ewing, Stoughton, Mass.; College of Physicians and Surgeons, Baltimore, 1897; member of the American Medical Association; served during World War I; associate medical examiner for the Norfolk County fifth district; formerly a member of the school committee in Stoughton; died March 8, aged 74.

LeRoy Ellis Fosner ✠ Evanston, Wyo.; Maryland Medical College, Baltimore, 1911; secretary of the Uinta County Medical Society; served as mayor, county health officer and county physician; died April 21, aged 61, of cerebral hemorrhage.

Edward Frank Freedman, Cleveland; Western Reserve University School of Medicine, Cleveland, 1913; specialist certified by the American Board of Radiology; member of the Radiological Society of North America; affiliated with the Mount Sinai Hospital, where he died April 16, aged 59, of coronary thrombosis.

Frederick Joseph Garlick ✠ Rochester, N. Y.; Albany Medical College, 1911; member of the American Urological Association; served overseas during World War I; consultant, Rochester State Hospital; served on the staff of the Monroe County Hospital; chief urologist, Park Avenue Hospital and the Rochester General Hospital, where he died April 22, aged 59, of arteriosclerotic heart disease.

Coleman P. Gay, Geneva, Ala.; Southern Medical College, Atlanta, 1897; member of the American Medical Association; past president of the Geneva County Medical Society; killed April 19, aged 73, when his automobile collided with a truck.

Lemuel B. Gilbert, McMinnville, Tenn.; Chattanooga (Tenn.) Medical College, 1903; died in the Rutherford Hospital, Murfreesboro, April 11, aged 72, of leukemia.

Herman Gliboff, Rockaway Beach, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1919; member of the American Medical Association; school physician; on the staffs of the St. Joseph Hospital in Far Rockaway and the Rockaway Beach Hospital; died in the New York Hospital April 13, aged 53, of carcinoma of the pancreas.

Rush Clayton Godfrey, Lancaster, Wis.; University of Pennsylvania School of Medicine, Philadelphia, 1912; member of the American Medical Association; past president of the Grant County Medical Society; served during World War I; physician in charge of the Grant County Asylum and medical director of the Lancaster General Hospital; died April 14, aged 61, of chronic myocarditis and arteriosclerosis.

Frank Goldenberg, Cincinnati; University of Cincinnati College of Medicine, 1914; on the staff of the Jewish Hospital; died April 16, aged 60, of coronary thrombosis.

Bernhard Alexander Goldmann ✠ Pittsburgh; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1922; assistant professor of dermatology at the University of Pittsburgh School of Medicine; specialist certified by the American Board of Dermatology and Syphilology; member of the American Academy of Dermatology and Syphilology; on the staffs of the Woman's Hospital and the Presbyterian Hospital, where he died April 7, aged 51, of coronary disease.

William T. Griggs, Poplar Branch, N. C.; University of Virginia Department of Medicine, Charlottesville, 1896; member of the American Medical Association; served as assistant surgeon in the U. S. Public Health Service; died April 9, aged 80, of arteriosclerosis and heart disease.

August Groman, Odebolt, Iowa; Chicago Homeopathic Medical College, 1878; member of the American Medical Association; died April 22, aged 90, of angina pectoris.

Malcolm C. Guthrie ✠ Kingston, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1907; formerly on the consultant staff of the Wilkes-Barre (Pa.) General Hospital; died April 14, aged 65, of heart disease.

John Franklin Habermel ✠ New Albany, Ind.; University of Louisville School of Medicine, 1928; fellow of the American College of Surgeons; member of the staff of St. Edward's Hospital; died in St. Joseph's Infirmary, Louisville, April 4, aged 47, of acute bacterial endocarditis.

Chris Richard Halloran ✠ Los Angeles; University of Texas School of Medicine, Galveston, 1923; member of the American Academy of Dermatology and Syphilology; for many years secretary of the Los Angeles Dermatological Society; died March 8, aged 50.

Robert Samuel Harwood ✠ Los Angeles; Chicago College of Medicine and Surgery, 1915; died in March, aged 58.

Forest Jacob Isenberger, Gypsum, Kan.; Kansas Medical College, Medical Department of Washburn College, Topeka, 1908; died in Topeka March 25, aged 78, of cerebral hemorrhage.

Lewis Turner Jackson, Tulsa, Okla.; St. Louis College of Physicians and Surgeons, 1906; died March 31, aged 69, of influenza.

George Howard Jantzen ✠ Queens Village, N. Y.; Fordham University School of Medicine, New York, 1920; examining physician for St. Anne Council, Knights of Columbus; on the staffs of the Queens General Hospital, Jamaica Hospital and the Mary Immaculate Hospital in Jamaica, where he died April 2, aged 51, of nephritis.

Frederick Henry Johanboeke, Louisville, Ky.; University of Louisville Medical Department, 1896; also a graduate in pharmacy; member of the American Medical Association; died in the Kentucky Baptist Hospital April 4, aged 86, of arteriosclerosis and chronic lymphatic leukemia.

Edward Samuel Johnston, Cincinnati; Cincinnati College of Medicine and Surgery, 1898; died April 20, aged 79, of acute congestive heart failure.

Frank Benedict Keleher, Brooklyn; Long Island College Hospital, Brooklyn, 1895; affiliated with St. Mary's Hospital, where he died April 18, aged 73, of perforated duodenal ulcer and general peritonitis.

Clarence Fay Kercheval, Clinton, Ind.; Illinois Medical College, Chicago, 1898; died April 2, aged 74, of chronic emphysema.

George Morris Kersey, Atlanta, Ga.; Meharry Medical College, Nashville, Tenn., 1940; interned at the Freedmen's Hospital in Washington, D. C.; served a residency at the Trinity Hospital in Detroit; a captain in the medical corps, Army of the United States, during World War II, serving as an associate medical officer at the Veterans Administration Facility in Tuskegee, Ala.; died April 16, aged 34, of dissecting aneurysm of the aorta.

Robert Kettner, Denver; University of Nashville (Tenn.) Medical Department, 1890; also a dentist; died in Albuquerque, N. M., March 1, aged 86, of cerebral hemorrhage.

Drue King, Tuskegee, Ala.; Tufts College Medical School, Boston, 1914; for many years affiliated with the Veterans Administration Hospital, where he died April 20, aged 60, of cerebral hemorrhage.

Russell Henry King, Pittsburgh; Western Pennsylvania Medical College, Pittsburgh, 1904; served during World War I; affiliated with the St. John's General Hospital; died April 24, aged 68, of coronary thrombosis.

Harold William Klewer, Toledo, Ohio; University of Cincinnati College of Medicine, 1935; member of the American Medical Association; interned at the Lucas County General Hospital; served in the medical corps of the U. S. Naval Reserve during World War II; on the staffs of the Maumee Valley Hospital, Toledo Hospital and the Mercy Hospital, where he died April 17, aged 40, of uremia.

Boyden Waclaw Kowalski ✠ Philadelphia; Hahnemann Medical College and Hospital of Philadelphia, 1928; honorary deputy sheriff of Ocean County, N. J., died April 9, aged 43, of carcinoma of the bronchus.

Frank George Kreft, Toledo, Ohio; University of Cincinnati College of Medicine, 1915; for many years county coroner; served during World War I; on the staff of St. Vincent's Hospital, where he died April 9, aged 56, of subarachnoid hemorrhage.

James Wesley Krichbaum, Montclair, N. J.; the Hahnemann Medical College and Hospital, Chicago, 1891; served on the faculty of the New York Homeopathic Medical College and Flower Hospital in New York; formerly on the staff of the Montclair Community Hospital; died April 22, aged 85, of cardiac decompensation and arteriosclerosis.

Charles Edward Lambert, New York; Bellevue Hospital Medical College, New York, 1893; died in St. Luke's Hospital April 6, aged 80, of arteriosclerosis and pneumonia.

Jennie Theodate Miner Lane, Worcester, Mass.; Boston University School of Medicine, 1896; died April 9, aged 86, of cerebral arteriosclerosis.

Damon Smith Latimer, Union City, Tenn.; University of Tennessee College of Medicine, Memphis, 1917; member of the American Medical Association; formerly secretary of the Obion County Medical Society; died in the Baptist Hospital, Memphis, April 26, aged 52, of cerebral hemorrhage.

William George List, Cincinnati; Medical College of Ohio, Cincinnati, 1896; member of the American Medical Association; at one time on the faculty of his alma mater; on the courtesy staff of the Jewish Hospital, where he died April 4, aged 71, of influenzal meningitis.

Philip Coe McDowell ✠ Wooster, Ohio; Western Reserve University School of Medicine, Cleveland, 1918; fellow of the American College of Surgeons; formerly in charge of the American Presbyterian Mission, Tehran Hospital, Tehran, Iran; died April 10, aged 55, of coronary thrombosis.

William Peter Martin ✠ South Orange, N. J.; Baltimore University School of Medicine, 1902; died in the Hospital of St. Barnabas in Newark April 2, aged 77, of chronic glomerulonephritis, diabetes mellitus and uremia.

Emery Allen Paschal, St. Charles, Ill.; Rush Medical College, Chicago, 1879; died in the Delnor Hospital April 2, aged 90, of pneumonia.

Frances L. Patrick, Chicago; Northwestern University Woman's Medical School, Chicago, 1894; died April 12, aged 86, of chronic myocarditis.

Theodora Garthwaite Tucker Peck, Elizabeth, N. J.; New York Medical College and Hospital for Women, Homeopathic, New York, 1900; died in the Richmond Memorial Hospital in Staten Island April 4, aged 69, of arteriosclerotic heart disease and pulmonary infarction.

Alexander C. Peska ✠ Chicago; Loyola University School of Medicine, Chicago, 1922; on the staff of the Lutheran Deaconess Hospital; died April 9, aged 58, of coronary occlusion.

Stephen J. Podlewski, Steubenville, Ohio; Baltimore Medical College, 1895; member of the American Medical Association; vice president of the Peoples National Bank; for many years surgeon for the Pennsylvania Railroad Company; on the staff of the Gill Memorial and Ohio Valley hospitals; died April 8, aged 74, of heart disease.

Adolf Hans Potthast ✠ Weimar, Texas; University of Texas School of Medicine, Galveston, 1915; physician for the Selective Service during World War I and II; served as city health officer; died in the Medical and Surgical Memorial Hospital in San Antonio, April 24, aged 57, of a malignant tumor.

Dominic Antonio Puleo, New York; Georgetown University School of Medicine, Washington, D. C., 1920; member of the American Medical Association; died April 19, aged 54.

Frederick Carver Robbins, Canandaigua, N. Y.; Boston University School of Medicine, 1896; member of the American Medical Association and the American Psychiatric Association; served during World War I; lieutenant colonel, medical reserve corps, not on active duty; at one time affiliated with the Cornell University in Ithaca; served on the staffs of Veterans Administration facilities in various places; on the staff of the Brigham Hall Hospital; died in Tampa, Fla., April 16, aged 73, of coronary occlusion.

W. Arch Robinson, Sturgeon, Mo.; Barnes Medical College, St. Louis, 1898; member of the American Medical Association; died April 10, aged 71, of myocarditis.

William Campbell Roland, Indianapolis; Kentucky School of Medicine, Louisville, 1893, veteran of the Spanish-American War and World War I; died in Fort Harrison, Ind., March 2, aged 78, of cerebral thrombosis and arteriosclerosis.

Edward Gould Rowland, Trenton, N. J.; Baltimore Medical College, 1903; specialist certified by the American Board of Psychiatry and Neurology, Inc.; member of the American Psychiatric Association; served on the staffs of the Norwich State Hospital in Norwich, Conn., Madison State Hospital in North Madison, Ind., and the New Jersey State Hospital; died in Philadelphia April 9, aged 68, of lymphosarcoma.

Abigail A. Shortess Ruston, Evansville, Ind.; Physio-Medical College of Indiana, Indianapolis, 1900; died April 28, aged 77, of hypertensive heart disease and hypostatic pneumonia.

Walter R. Shannon ✠ Milford, Pa.; Baltimore University School of Medicine, 1897; past president of the Wayne-Pike Counties Medical Society; county medical director; examining physician for the county Selective Service during World War II; on the courtesy staff of St. Francis Hospital in Port Jervis, N. Y.; at one time surgeon for the Erie Railroad and a member of the state legislature; died in Allentown April 7, aged 71, of coronary occlusion.

William Henry Shields, Ellwood City, Pa.; Medico-Chirurgical College of Philadelphia, 1893; on the staff of the Ellwood City Hospital; died in the Shadyside Hospital in Pittsburgh April 24, aged 78, of coronary occlusion, hypertension and cerebral hemorrhage.

Mayer Shoyer, Holton, Kan.; University Medical College of Kansas City, Mo., 1897; member of the American Medical Association; county health officer; affiliated with St. Francis Hospital in Topeka; died April 5, aged 71, of sarcoma of the liver.

Elijah W. Smith, Benton Harbor, Mich.; Indiana Eclectic Medical College, Indianapolis, 1890; Barnes Medical College, St. Louis, 1893; formerly associated with the Indian Service; died March 30, aged 89, of pneumonia.

Carlos C. Stevens ✠ Blytheville, Ark.; University of Georgia Medical Department, Augusta, 1902; on the staff of the Blytheville City Hospital, where he died March 23, aged 68, of heart disease.

Jesse Oldham Strother ✠ Henderson, Ky.; Kentucky University Medical Department, Louisville, 1905; on the staff of the Deaconess Hospital in Evansville, Ind.; member and past president of the staff of the Henderson Hospital, where he died April 21, aged 65, of coronary thrombosis.

Reginald James Henry Stroud ✠ Tempe, Ariz.; University of Colorado School of Medicine, Denver, 1912; served as secretary and state superintendent of public health; member of the House of Delegates of the American Medical Association from 1919 to 1930; formerly affiliated with the Indian Service; past president of the Maricopa County Medical Society and the Cochise County Medical Society; commissioner for the Amateur Athletic Union; formerly affiliated with the Cochise County Hospital at Douglas; served on the staffs of the Southside District Hospital in Mesa, St. Joseph Hospital and Good Samaritan Hospital in Phoenix, where he died April 4, aged 60, of cholelithiasis, hepatitis and diabetes mellitus.

Anna E. Daniels Trimble, Middleport, Ohio; Cincinnati College of Medicine and Surgery, 1896; died March 12, aged 78, of cerebral embolism.

Thomas Francis Welch, Washington, D. C.; Georgetown University School of Medicine, Washington, 1922; interned at the Central Dispensary and Emergency Hospital; died in Towson, Md., April 5, aged 48, of pulmonary tuberculosis.

Frank Moore Whiting, Oroville, Calif.; Cooper Medical College, San Francisco, 1906; served during World War I; deputy county health officer; died April 26, aged 71, of carcinoma.

Creed Scott Wilkins, Abbeville, Miss.; Memphis (Tenn.) Hospital Medical College, 1911; director of the First National Bank of Oxford; died March 16, aged 59, of heart disease.

James Edgar Wilson, Lancaster, Texas; University of Texas School of Medicine, Galveston, 1896; at one time assistant health officer of Dallas; served during World War I; during World War II examining physician for the local draft board; served on the local school board; died March 8, aged 73, of coronary occlusion.

William Francis Wood, Bedford, Va.; College of Physicians and Surgeons, Baltimore, 1893; died April 19, aged 84, of myocarditis.

Daniel Yellin, San Francisco; University of California Medical School, San Francisco, 1939; member of the American Medical Association; affiliated with the Mount Zion Hospital, where he died March 5, aged 32, of lupus erythematosus.

Foreign Letters

LONDON

(From Our Regular Correspondent)

June 14, 1947.

First Step in Organizing the National Health Service

The National Health Service is now law, but the organization of a medical service for every person, which includes every form of treatment, general practitioner, consultant and hospital, is a gigantic task which has only begun. As a first step the executive councils regulations have been laid before Parliament by the minister of health. The act envisages the setting up of executive councils in each county and county borough to organize the family practitioner services locally. The regulations deal with arrangements for appointing members, terms of office, and general procedure. Half the membership of the councils is to be professional and appointed by the medical profession itself, and half is to be appointed by the local health authorities and the minister. The regulations provide for the annual retirement of one third of the members and thus set up the normal term of office as three years. Most of the appointments to the first executive councils have already been made by the local authorities and the local medical organizations, and the minister of health is now considering what appointments he will make.

The executive councils will not only have to begin planning the services for which they will themselves be responsible but they will also have to give any advice or help sought by bodies responsible for organizing other parts of the service, such as local health authorities and the regional hospital boards. The regulations also provide for the appointment by these councils of ophthalmic services committees which will organize the local supplementary eye service, which is to be provided by physicians with prescribed qualifications and by ophthalmic and dispensing opticians. It is hoped that the health service will come into operation next April.

Poisoning by Dried Egg

During the war the importation of spray dried egg was a valuable means of economizing shipping space. But a number of cases of poisoning due to Salmonella organisms occurred. This has led the Medical Research Council to make an investigation, the result of which is published in a pamphlet entitled "The Bacteriology of Spray Dried Egg, with Special Reference to Food Poisoning." Examination of 7,584 samples of dried egg showed the presence of Salmonella organisms in 9.9 per cent. In 1944 the number of known outbreaks of food poisoning due to Salmonella reached the unprecedented total of 454, a figure greater than that for seventeen years preceding the war.

Between 1942 and the end of 1945, 244,000 tons of dried eggs were imported from the United States, Canada and the Argentine. Though the risk of food poisoning was apparent, consumption continued because of the high value as a concentrated foodstuff. Dishes of dried eggs were prepared in the experimental kitchen of the Ministry of Food and tested at the bacteriologic laboratory at Cambridge. It was proved that dried egg was safe to eat if cooked soon after reconstitution, even by such a relatively mild heat treatment as scrambling. Recipes for custard sauces prepared without boiling and milk and egg shakes were rejected.

Gift for Repair of the Royal College of Surgeons

A gift of $40,000 from the American College of Surgeons has been received by the president of the Royal College of Surgeons as a contribution to the fund for the restoration of the Royal College so extensively damaged in 1941 by German bombs. This gift brings the total so far received to $700,000 toward the $1,000,000 required. The American College is also giving a desk and lantern for the lecture theater of the Royal College.

To Reintroduce Bill to Legalize Euthanasia

The Voluntary Euthanasia Society, which introduced a bill in the House of Lords to legalize euthanasia for patients suffering intolerable pain from incurable disease, has held its annual meeting. The bill was presented in 1936 but failed to secure a second reading. At the meeting Dr. E. A. Barton, who had retired after forty years' practice in London, said that doctors who acceded to a patient's appeal for release took a risk. Though cases needing euthanasia are rare—he had seen only half a dozen in fifty years—they were peremptory. He hoped that many would be willing to risk criticism and worse by doing for the patient what they would wish to be done for themselves in similar circumstances. The society proposes to reintroduce the bill. It is suggested that England should be divided into areas, in each of which two doctors of high standing should be appointed who could be called on by the general practitioner to carry out the final act. The patient would have to ask for the relief, but only urgent pleas would be considered. The consent of the friends would not be asked, for, if refused, the patient must continue to suffer, and if it was given, misunderstandings might arise later. Though euthanasia is not legal, the authorities do not seem disposed to take any action in the cases in which relief has been given.

Sir Almroth Wright

The death of Sir Almroth Wright at the age of 85 has removed from medicine a great and unique figure. The son of an Irish father and Swedish mother, he was educated at Trinity College, Dublin, where he graduated B.A. in 1882 and won the gold medal in modern literature. He had already taken up medicine, in which he graduated in 1883. His pioneer work on immunization began in 1896. Inspired by Haffkine's inoculation of cases of cholera and Pfeiffer's inoculation of 2 cases of typhoid, Wright introduced the idea of immunization by vaccines made of killed bacteria. Pasteur had obtained results by inoculation of living but attenuated cultures. Wright, however, thought that inoculation of living microbes to produce immunity in man would not be practicable on a large scale. He therefore perfected the technic of inoculation of killed typhoid cultures and showed that they would protect against the disease. He then came up against the opposition of the army authorities, whose conservatism could not stomach the novel idea of introducing microbes, dead or alive, into the human body. So powerful was the opposition that he had to resign his army post. However, a supply of his vaccine was dispatched with the troops sent to South Africa to fight the Boers. There, as in other campaigns, typhoid was rife, owing to insanitary conditions in a dusty climate, which led to a plague of flies as carriers. The results were most satisfactory. Wright was able to show that at Ladysmith only 2 per cent of 1,705 men who had been inoculated contracted typhoid, while 13.2 per cent of those not inoculated did. A similar result was obtained in troops stationed in India. The result was that the War Office adopted typhoid inoculation as a routine procedure.

In 1902 Wright obtained his most important appointment, which he held nearly to the end of his life—pathologist to St. Mary's Hospital, London. Under his direction a special department was created, the Department of Therapeutic inoculation, which afterward became the Institute of Pathology and Research. The institute provided sufficient antityphoid vaccine for 4½ million men. His work centered on the possibility of controlling bacterial diseases already in progress by inoculation

of killed cultures (vaccines). He was not content to measure the results clinically. He examined the patient's blood for indications that the body had responded by the production of protective substances. This led to new technical procedures for the measurement of phagocytosis and bactericidal power, which he embodied in a book published in 1912 entitled "Technique of the Teat and the Capillary Glass Tube." It was an important first step in the application of micro-technic to bacteriology.

Sir Frederick Gowland Hopkins

The death of Sir Frederick Gowland Hopkins in his eighty-sixth year has removed the man who played the greatest part in the creation of the science of biochemistry. He is most widely known as the discoverer of the vitamins, but his other fundamental research included the part played by lactic acid in muscle activity, the discovery of the amino acid tryptophan and its "specific biologic value," and the isolation of the cell catalyst glutathione. His scientific life began with the study of chemistry. The government analyst and medical jurist Sir Thomas Stevenson appointed Hopkins his assistant. He showed great skill in the isolation and identification of poisons in famous criminal cases. He next studied medicine and qualified but never practiced. His chemical training directed him toward pathologic research. In 1896 he was largely instrumental in the founding of the Clinical Research Association, an entirely new kind of undertaking which examined pathologic specimens for physicians and reported on them. Toward the close of the century came a turning point in his career. He attracted the notice of the physiologist Sir Michael Foster, who occupied the chair at Cambridge and invited Hopkins to go there and develop teaching and research in the chemical side of physiology. He was appointed lecturer in chemical physiology and had not been long at Cambridge when, in 1901, he made his first great discovery. In collaboration with S. W. Cole he isolated the amino acid tryptophan. In 1914 a professorship in biochemistry was created for him. In his later years he took an active part in the direction of scientific research. He was an original member of the Medical Research Council. In 1931 he was elected president of the Royal Society, the highest scientific position in this country. Under him the Cambridge School of Biochemistry became famous for investigations into the chemistry of life. Visitors came to his laboratory from all over the globe and went away impressed by the man and his work.

PARIS

(From Our Regular Correspondent)

July 6, 1947.

Poliomyelitis in France

The following figures show that poliomyelitis has lately attained importance among infectious diseases in France:

Year	Months	Number of Cases
1943	June to November	1,800
1944	June to November	366
1945	June to November	780
1946	June to November	1,342

Two small epidemics are to be noted during the years 1943 and 1946. In the period from the 1st of January to the 1st of May 1947, 182 cases have been recorded, and the Bulletin of Epidemiologic Information of the Ministry for Public Health states that "these figures surpass the average and remain close to the maximum of cases noted in the course of ten years."

The 1943 Epidemic

This epidemic began at Montluçon and had two foci, one in the Allier and Haute-Garonne departments, the other in the Meurthe-and-Moselle department. Its chief characteristics were that (1) in addition to 1 to 6 year old children a number of adults and adolescents were stricken, (2) cases with early meningeal symptoms were rare and (3) the paraplegic and monoplegic forms were prevalent (65 per cent). The mortality rate was 10.5 per cent.

The 1946 Epidemic

This epidemic produced 51 cases in June, 155 cases in July, 335 cases in August, 376 cases in September, 278 cases in October and 127 cases in November. It had one focus in the department of Maine-et-Loire and, generally speaking, in the regions bordering on the Loire River, between Saumur and Angers, and another focus in the Côte d'Or, in the Beaune district. As in 1943, the epidemic did not affect Paris. All cases reported for Paris have been sporadic or those of adults and children, who appear to have contracted the disease in the other infected areas or who had been in contact with infected persons and came back to Paris during the incubation period. While the river Seine cannot be considered as a source of contagion, nevertheless the prohibition of bathing in rivers was one of the first prophylactic measures taken. The epidemic was characterized by the existence of typical paralytic cases, but there were also atypical cases with meningeal or encephalic syndromes. Paralleling this poliomyelitis epidemic, encephalitis was noted in horses, as well as other epidemics in chickens. All efforts, including laboratory studies of those animal diseases, made with a view of discovering the origin of poliomyelitis, have given no result.

At several meetings of the National Academy of Medicine, Debré, Lemierre and Levaditi discussed the means of transmission of poliomyelitis. Debré and Lemierre voiced doubts regarding its propagation by the enteral route, but Levaditi upheld this theory. In discussing respiratory failure in poliomyelitis, Debré emphasized that, in the case of serious paralysis of the respiratory muscles, the prognosis does not improve as a result of artificial respiration provided in the steel lung.

Sanitary Condition in the Overseas Territories

A special meeting April 29 of the National Academy of Medicine, under the presidency of Mr. Jolly, was devoted to exotic pathology. General A. Sice, inspector of colonial troops, presented a survey of the present health condition of French overseas territories. Thanks to the efforts of the Service of General Hygiene, improvement in the cultivation of foodstuffs has taken place. A stubborn fight against the state of underfeeding of the population has increased the resistance of the people against infections by restoring alimentary equilibrium. As a consequence a decrease in epidemics, once murderous, has been recorded. Smallpox has been kept in check everywhere. In 1943 a local epidemic brought by nomads made its appearance at Fort Lamy; the situation was straightened out after 626,873 smallpox vaccinations and 78,921 associated vaccinations with a vaccine produced in the vaccination center of Fort Lamy, under control of the Brazzaville Pasteur Institute, had been effected. Human trypanosomiasis, which formerly destroyed the population of entire villages, is regressing. In French West Africa, out of 4,500,000 inhabitants examined, 300,000 were found ill and treated. In French Equatorial Africa,–out of a population of 3,800,000 inhabitants, 1,472,700 were examined, among whom 7,600 were found ill. Paludism, especially in Madagascar and in La Réunion Island, is not yet checked. In 1945 Madagascar alone had 777,000 cases. Leprosy is progressing, on account of the lack of hygiene, in the zones where the disease is endemic. Plague is regressing and the number of cases, which was 681 in 1939, was 184 in 1945; 3,000,000 vaccinations were performed with the living vaccine prepared by Girard at the Tananarive Pasteur Institute.

Yellow fever is also decreasing. In West Africa, 23 cases were recorded in 1941; there were only 2 in 1944. Since 1941, in Free French Africa, anti-yellow fever vaccination has been declared compulsory. The number of persons subjected to vaccination increased from 8,000,000 in 1941 to 15,000,000 in 1945. Syphilis still exerts its nefarious influence on infantile mortality. As to the experimental work of the branches of the Pasteur Institute, especially to be noted is the importance of a polyvalent anti-snake bite serum of the Brazzaville Pasteur Institute. This is very effective against the venoms of Naja melanoleuka and of Dendropsis jameson. A permanent commission for anthropologic studies of the aboriginees of Africa has been created to study their migrations and their intermixtures.

ITALY

(From Our Regular Correspondent)

June 5, 1947.

National Surgical Congress

The first postwar national surgical congress was held in Florence at the same time as that for internal medicine. Professor Pedotti, chairman of the Swiss Surgical Society, and Professor Datymer, representing the surgeons of Poland, participated.

The first joint session with the medical association was devoted to papers on tumors of the colon. Professor Alessandrini of the University of Rome presented a paper from the medical point of view, Professor Matronola of Rome from the surgical point of view, and Professor Cignolini of Genoa from the roentgenologic point of view.

Professor Alessandrini's paper was devoted particularly to epitheliomas of the colon and to their early diagnosis; he emphasized the progressive increase of these neoplasms as demonstrated by clinical and anatomicopathologic statistics. According to the speaker the continued use of laxatives with liquid petrolatum as a base or of derivatives of anthracene or phenanthrene for years or decades may be considered as one of the causes of the increase. With regard to therapy, surgical intervention may be the method of choice as soon as the diagnosis of epithelioma of the colon has been established; it should be preceded by adequate medical preparation (rational irradiation, insulin therapy, occasionally blood transfusions, sulfonamide therapy). The opportunity of surgical intervention for diffuse polyposis is controversial. A substantial diet is suggested for inoperable cases; it should be poor in cellulose, in fats and in sodium chloride but rich in potassium.

In discussing surgical intervention, Professor Matronola stated that the first step should be exploratory, consisting in an extensive laparotomy which allows for examination of the internal abdominal cavity. The location and the extent of the tumor should be established on exploration as well as the occurrence of adjacent or distant metastases, and it should be decided whether the removal of the tumor will be feasible. In case it should not be feasible, one may resort to a palliative operation, i. e. an internal derivation (anastomosis) or an external one (artificial anus). Hemicolectomy may be performed as a radical operation for cancer of the right colon, but there is no agreement with regard to one or two stages. According to the speaker, intervention in two stages may be generally preferred. There is no consensus with regard to the method of choice for intervention of cancer of the transverse colon: segmentary resection, the Mikulicz operation, hemicolectomy. The mortality rate is high. More difficult problems may be presented to the surgeon by the left colon. For cancer of the left angle and of the descending colon, left hemicolectomy may be preferred in most cases, preceded by a cecal anus and followed by transverse sigmoidostomy. Cancers of the sigmoid flexure may easily be extirpated because of the length of the mesentery. The simplest and safest method of treatment of epithelioma of the rectosigmoid consists in low amputation, which may be terminated by a definite iliac anus. The operative mortality rate has been reduced to a negligible percentage.

Professor Cignolini demonstrated the great difficulties met on x-ray examination of the colon. He stressed the notable parallelism between the neoplastic symptomatology of the colon and that of the stomach. The greatest difficulty in establishing a differential diagnosis between intestinal and gastric forms results from the fact that the pathology of the colon is much more variable than that of the stomach. With the aid of the technic of intestinal intubation it becomes possible in many cases to localize and to define the obstructional lesion.

Trauma of the Thorax

Professor Gherardo Forni of the University of Bologna demonstrated that the thoracic viscera may be exposed to violent and highly complex traumatism because of their plurality and their vital functions. The symptoms vary according to the anatomic lesion and the intensity of the trauma. Pain may be predominant in parietal contusions with skeletal lesions and may be exacerbated with respiration. Pulmonary and cardiac lesions may be manifested by cough, blood in the sputum, dyspnea, cyanosis, tachycardia and arrhythmia. X-ray examination is of great significance for demonstrating a displacement of the viscera, the loss of their normal relationship and the occurrence of fluid or gas in the pleural and pericardial cavities. Surgical intervention may be required by the occurrence of a hypertensive pneumothorax, of a generalized emphysema spreading to the mediastinum or by a profuse hemothorax. It presents excellent therapeutic judgment in case of profuse and threatening hemothorax to remove the blood and to induce a pneumothorax (Morelli). A pneumothorax of mild degree may not require any treatment; in pneumothorax with high tension, puncture may be performed. Bilateral pneumothorax, which presents a serious threat to the life of the patient, requires immediate intervention: puncture of the thorax and occasionally thoracotomy with suture of pulmonary lacerations. In generalized emphysema with cyanosis and progressive asphyxia a suprasternal incision may be made and an aperture of the mediastinum with application of drainage by means of a cupping glass.

Cancer of the Uterus

Professor Antonio Biancheri believes that subtotal hysterectomy may be the treatment of choice in all cases in which removal of the uterus may be required for fibroma or for connected causes in the absence of any involvement of the cervix. In those cases, however, in which the cervix presents lesions from which suspicions of a precancerous condition may arise it may be necessary to remove the mucosa or the involved segment of the cervix by means of electrocoagulation or by the application of radium. In cancer associated with pregnancy, Wertheim's operation remains the method of choice according to the statistics of Italian gynecologists.

Professor Luigi Caravani believes that the symptoms which had been considered as an initial manifestation of cancer of the body of the uterus should be interpreted as one of a spreading process of the already present blastomatous permeation of the entire female genital system. Consequently he suggested total colpohysterectomy by the abdominoparavaginal route, with the removal of the entire genital complex and all the glands connected with it. The speaker contrasted the high postoperative mortality rate (actually about 28 per cent, but with a possibility of being reduced with improved technic) with the high percentage (better than 80) of permanent recovery in women with cancer who received similar treatment.

Professor Eugenio Milani emphasized that the guiding line for radiologic treatment of cancer of the cervix of the uterus should be in general that endocavitary Curie therapy may be combined with transcutaneous roentgenotherapy. A two stage treatment may be effective in the majority of cases, but there are also workers who favor simultaneous treatment. A combined treatment may likewise be the guiding line for radiologic treatment of the body of the uterus in all the inoperable cases as well as in those that have passed the first stage. For the operable cases the statistics demonstrate that the percentage of a five to ten year recovery may be higher if surgical intervention can be combined with postoperative roentgenotherapy. The treatment of recurrence and metastasis may be essentially radiologic.

Executive Committee

The new executive committee of the Italian Surgical Society was elected with Professor Luigi Torraca as president.

The subjects for papers at the next congress are (1) surgical treatment of pulmonary tuberculosis, (2) surgical treatment and treatment with estrus producing compounds for cancer of the prostate, (3) cicatricial stenosis of the esophagus and (4) (in cooperation with the Italian Society of Internal Medicine) mediastinal syndromes.

MOSCOW

(From Our Regular Correspondent)

June 7, 1947.

Antituberculosis Vaccination

The work of Soviet scientists begun in 1925 demonstrated that Calmette's antituberculosis vaccine decreases the mortality among vaccinated children from two to four times. Those figures are based on prewar data when about three million children were vaccinated—more than in all other countries of the world. Weisfeiler and his collaborators demonstrated that Calmette-Guérin bacillus efficiency during parenteral administration is much higher when used twice. Professor Pokrovskaya showed the possibility of vaccine intensification by means of a nonspecific mesenchymotropic preparation. Khmelnitskaya and Rabinovich found that small doses of Calmette-Guérin bacillus used subcutaneously have in certain types of tuberculosis a curative effect. Weisfeiler, Baydakov and Tourgetsky could not confirm Calmette's observations on the rapid destruction of their bacillus in the vaccine fluid. If it is obtained from young cultures, its immunizing properties are preserved about one month. In 1937 Weisfeiler reported on obtaining a dry vaccine with a longer expiration date. His collaborator Leschinskaya worked out a method of preparing a dry glucose vaccine, and it has been used since 1943, when the people of distant areas and regions released from fascist occupation were immunized with the dry glucose vaccine. Great Britain has begun experiments with the dry vaccine, based on the Soviet method.

At present methods for obtaining a more potent vaccine are being worked out, and a search is being made for new cultures with high immunizing properties. Exact experimental figures are being collected on the grade of high quality dry vaccine and on optimal conservation conditions for the Calmette-Guérin bacillus in dry form.

Acclimatization of Residents in the Arctic Regions

The aim of the expedition in the arctic region of two Institutes of the Academy of Medical Sciences of the U. S. S. R.—General Hygiene and Nutrition, in 1946, was a mass observation of the population. It was noted that inhabitants of Northern regions, become accustomed to a new environment best of all—those from middle and central parts of the Soviet Union do so with some difficulty—and natives of the South do so worst of all. In this climate the time of work is changed four times yearly because of the long polar night. Nearly 80 per cent of the residents after arriving suffer from nostalgia, which completely disappears when people are busy working.

The morbidity in the arctic is less than in the other parts of the Soviet Union. Living in conditions of eternal frost results in freezing of the feet even during summer because the soil layer (exposed to melting) does not exceed 40 cm. Wounds close without suppuration because of the sterility of the air and small virulence of micro-organisms. Comparatively frequent is appendicitis. Arthralgia occurs with the low temperature but without objective changes in the joints. Heart and blood vessel diseases are quite rare. Caries is very common; it spreads quickly and is accompanied with pyorrhea. One of its possible causes is the use of demineralized snow water.

The investigators find that the arctic climate will have a favorable influence. The polar night is characterized by many factors having a stimulating influence—electrical status of the atmosphere and presence of enormous quantities of ionized air rich with ozone. The polar radiation also is stimulating. The first objective is to organize a work and rest regimen in order to maintain a normal physiologic rhythm.

Again this year, expeditions of scientific institutes in the arctic region are taking place to study the mechanism of the acclimatization processes.

Visceral Leishmaniasis (Kala-Azar)

Nicholas I. Latichev of the Institute of Epidemiology and Infectious Diseases of the Academy of Medical Sciences of the U. S. S. R. has reported a study of visceral leishmaniasis. In 1940 in Middle Asia about 2,000 cases were identified and in the Precaucasus about 500. Thus kala-azar takes an insignificant place among other diseases, in our country, especially in comparison with India, where it sometimes takes on the character of an epidemic.

During the building works in 1933 in the valley of the Waczch River (South Tajikistan) there were a number of cases of "chinidine resistant malaria without parasites in the blood," which were later identified as visceral leishmaniasis. When the epidemiology of the disease was studied the jackal was suspected as a reservoir of the virus and it became necessary to demonstrate the existence of spontaneous infection in those animals. In 1946 in the liver and spleen of some jackals killed in the lower parts of Kafirjagan River Leishmania was identified. On the basis of these facts Latichev finds that visceral leishmaniasis is not a simple infection but has three different types. The first is a zoonosis affecting rural populations which are infected from natural reservoirs of the virus—jackals and stray dogs. The second type, anthroponosis, is found in towns where the principal source of infection is the patient and dogs are only a minor factor. The cause of the third type, Indian kala-azar, is closely connected with the human organism.

Institute of Gynecology and Obstetrics

At the third session of the Institute of Gynecology and Obstetrics of the Academy of Medical Sciences of the U. S. S. R. Professor E. Berkovich and his associates reported a study of placental substances influencing the mechanism of labor. They found that the placenta contains a large amount of acetylcholin which the placenta synthesizes. Its action is accelerated by vitamin B_1 and pituitary, which sensitize the uterine muscle. However, when verifying the action on the uterus of potassium, calcium, thiamine and pituitary it was demonstrated that its oxytocic effect is rather small. Thus the authors conclude that the blood oxytocic properties of lying-in women depend on acetylcholine circulating in their blood and diminution of cholinesterase.

Professor G. Salgannic and his associates spoke on the excitability of sensory centers of pregnant women. An increase of

cerebral excitability of the optical centers at the sixth month and one to two weeks ante partum was observed. During labor excitability of the cerebral sensory centers is decreased and soon after delivery reaches its usual figures. For evaluating the status of the vegetative, nervous and reticuloendothelial systems much clinical material was studied. During the second period of pregnancy and labor sympathicotonic symptoms are present; simultaneously the reticuloendothelium is depressed; only in certain cases can a total vegetative dystonia be observed. These observations demonstrate the influence of the protective systems in pregnant women and may be of use for increasing their resistance to infectious diseases.

The anesthetic and labor accelerating effect of thiamine reported by Professor R. Schub (THE JOURNAL, January 11, p. 122) was confirmed by dozent D. Ginodman and Drs. M. Tyurin and M. Ziroulnikov. They noted a parallelism between the content of vitamin B_1 in the organism, the duration of labor and labor pains. They were more frequent and powerful after thiamine administration. However, its anesthetic action is small.

MADRID

(From Our Regular Correspondent)

June 7, 1947.

Studies of the Spanish School of Cajal

The Spanish School of Histology and Pathology, which under S. Ramon y Cajal has become the pride of Spain, is still active in spite of all difficulties. The names of Pío del Río Hortega (who died a year ago) and J. F. Tello are well known and some of their recent work merits description. Tello, Cajal's favorite pupil, is working in his old laboratory at the Cajal Institute (now incorporated in the Higher Council for Scientific Research and subsidized by the state) and, since 1942, has carried out studies on the structure of the tactile and motor nerve endings.

As a result of his studies on the motor endings of batrachians and reptiles he has been able to prove that in all animals examined the ramification of the axon in the motor endings of the voluntary muscles appears enveloped in a nucleated sheath, which seems to be the continuation of the sheath of Schwann. More recently he has published two studies on neurogenesis. In the first he reviews the genesis of the sympathicus by Bielschowsky's method in blocks and not only confirms his previous ideas on mesodermic origin but sees this forming at the expense of syncytial cords of endothelium, starting from the aorta and resembling vascular buds.

Of special interest, because it is an original and fundamental contribution to knowledge of the disputed problem of the neurogenesis of cranial ganglions, especially of the part played in them by the neural crest, is his latest published work. In it he deals with the respective participation of the neural crest and the placodes in the formation of the sensitive cranial nerves (in chickens), proving that most, if not all, of the nervous elements proceed from the ectodermic placodes subsequent to the disappearance of the crest. All these papers were published in the review *Trabajos del Instituto Cajal de Investigaciones Biológicas* between 1942 and 1946.

Importance also attaches to the work of Dr. F. de Castro, professor in Seville University, carried out in the Cajal Institute (Madrid). Continuing his investigations by his crossed anastomosis method, which makes it possible to model a simple bineuronal reflex arc, he has arrived at a new concept of the synapsis in centers and peripheral ganglions. In the modeling of the synapsis, three discontinuous active elements are concerned without any trophic continuity between them: the affector element, the effector element and the intermediary element (neuroglia), whose protoplasm, interposed between the other two, serves as an indispensable mediator of the nervous current. This is sometimes the Schwann cell, sometimes the intercapsular cell of the ganglion and sometimes again the oligodendroglia itself. The genetic and morphologic identity of these elements, already upheld by Pío del Río Hortega and demonstrated by J. M. Ortiz Picon in the case of the ganglion cells, is verified by Rodriguez Perez in the case of the Schwann cell.

The doctrine of these Spanish authors is summed up in the concept of "tripolar synapsis," which offers a physiologic explanation of the polarization of current and the inhibition of the antidromic conduction at the level of the first intercalary station. These facts reinforce the thesis of the anatomic unity of the nerve cell.

Professor J. Sanz Ibañez, professor of histology and pathology in Madrid University, has carried out research on the experimental histopathology of poliomyelitis by Nissl's method and silver saturation. He studies the spinal medulla of mice, guinea pigs and monkeys with paralytic manifestations after virus infections and proves the existence of lesions of the central nervous system, above all in monkeys. But the fundamental change occurs in the peripheral nerves of the paralytic limbs (in the distal parts) and also in the myoneuronal synapsis, the thick nerve trunks being free from lesions. There are sometimes regenerative phenomena in the synapsis which, according to him, are evidence of the absence of central lesions, since that would not be possible if the spinal neuron was infected.

According to Sanz Ibañez the paralysis is of peripheral origin and is related to the motor plate, as is proved by the presence of viruses in the muscles of the paralytic legs of mice. He also studied the degenerative lesions of the liver, spleen, heart, kidneys, intestine and adrenals, which he attributes to the presence of virus in the blood. He describes clinical forms without paralysis or any neuraxial lesions and succeeds in adapting to guinea pigs the mouse virus by transmitting it in series and distinguishing (1) central paralytic forms, which are incurable, with lesion of the motor neurons; (2) peripheral paralytic forms, which are sometimes curable, since they affect only the neuromuscular synapsis; (3) nonparalytic forms with a febrile type (neutralization of the virus by the sick animal's plasma and not by the normal ones); (4) an apyretic nonparalytic type of great epidemiologic interest, since it might be a source for the transmission of the disease.

Dr. L. Arteta, head of the Necropsy Department of the Madrid General Hospital and Assistant Professor of the Faculty of Medicine, a pupil of the Cajal-Tello-De Castro trio, has made a deep study of pellagra. He has studied in great detail the tongues of pellagrous patients, microscopically and macroscopically, rigorously excluding possible causes of error, proving the existence of a noninflammatory (i. e. degenerative) segmentary neuritis with trophic alterations of the remaining components of the tongue. Basing his work on histology, he comes to the conclusion that glossitis is a disturbance consecutive to primitive alterations of the nervous system. Arteta proposes, to designate the absence of inflammatory manifestations and to honor the Spanish investigators who discovered pellagra, the use of the term "Casal's glossopathy."

Arteta also has described the technic of a new silver staining method which is particularly constant and applicable to large sections of organs of the nervous system preserved for a long time in solution of formaldehyde. Finally he has devised a technical modification of Ferry's method destined to make permanent the dryings with polychrome blue which so far have been used in the rapid diagnosis of pathologic tissues in surgical operations; he has achieved stabilization of the color, using a fixing solution of ammonium molybdate and a few devices of his own.

Correspondence

CARBOHYDRATE METABOLISM IN ULCER PATIENTS

To the Editor:—In their article "Gastric Operations" in THE JOURNAL, June 14, Zollinger and Hoerr describe a group of ulcer patients with postoperative symptoms which they attribute to a disturbance in blood sugar levels. One is led to believe that this disturbance is due to the altered gastrointestinal physiology caused by the surgery. These authors report that both hypoglycemic and hyperglycemic curves were observed in their patients. I studied sugar tolerance curves in a group of ulcer patients who had never undergone surgery and who were being treated medically in an outpatient department and in the ward. Practically every patient had an abnormal tolerance curve. The majority showed hypoglycemia, the remainder hyperglycemia. I also found similar abnormal curves in patients with typical ulcer symptoms but without signs of ulcer on x-ray examination. Both ulcer and pseudoulcer patients responded to a low carbohydrate diet aimed to overcome the abnormal blood sugar levels. The results of this study were published in a paper titled "The Pain Mechanism in Ulcer and Pseudoulcer Syndromes" (*Rev. Gastroenterol.* **7**:485 [March-April] 1940). There was a definite correlation between the pain in ulcer and a fall in blood sugar. In hypoglycemic patients the pain coincided with a fall to absolute hypoglycemic levels. In patients with hyperglycemia the pain coincided with periods of sharp fall, not necessarily to hypoglycemic levels. In hyperglycemic patients the fall in blood sugar was sharp and to "relative hypoglycemia," a term used by several authors to explain typical hypoglycemic symptoms in hyperglycemic patients. The relief from pain which follows the ingestion of food in ulcer was correlated with a rise in blood sugar following the ingestion of food.

From my studies I concluded that the fundamental cause of ulcer and pseudoulcer symptoms was a disturbance in carbohydrate metabolism caused primarily by the ingestion of foods containing starch and sugar. The relief from symptoms such as, pain, flatulence, heartburn, nausea, weakness, faintness, headache and syncope, with a low carbohydrate diet, was attributed to the normal blood sugar levels that are obtained with such a diet. It is thus apparent that the disturbance in carbohydrate metabolism in ulcer patients is fundamental and is present in the patient who has not been operated on. Surgery may aggravate this disturbance. The beneficial effects that are observed with frequent feedings in these patients I have attributed to the stabilizing effect of such a regimen on blood sugar levels. Frequent feedings of milk and cream are especially beneficial, since such a regimen contains no starch and little sugar.

The diet employed by me ranged from 2,300 to 2,900 calories (carbohydrate, 114 to 124 Gm.; protein, 101 to 129 Gm.; fat, 161 to 212 Gm.). Aside from six thin soda crackers each day, there was no starch or sugar allowed. Recently I have eliminated the crackers from the diet. The carbohydrate allowed was ingested chiefly in the form of 5 and 10 per cent vegetable. Fruits were allowed rarely, and no fruit juices were allowed at all. In recent years several authors have stressed the beneficial effects of protein foods and protein hydrolysates in the treatment of ulcer. These authors give one the impression that the protein is chiefly responsible for the good results obtained. This could apply in patients with hypoproteinemia. However, most ulcer patients have no hypoproteinemia. The diet employed in my study had a high protein content. But I have attributed the good results mainly to the absence of starch and sugar. A diet which is high in protein and which contains ordinary amounts of starch and sugar will not prove beneficial. The starch and sugar must be eliminated.

BENJAMIN P. SANDLER, M.D.,
Veterans Hospital, Sampson, N. Y.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Selective Service Act: Physician Need Not Reemploy Discharged Attorney to Handle Collection Work.—The plaintiff filed an action against the defendant physician claiming that he had been denied the reinstatement and compensation to which the provisions of the Selective Service Act entitled him. The case was heard in district court D, Maryland.

The defendant physician specialized in the examination and treatment of the eye and the prescribing and furnishing of eye glasses to his patients. Most of his large professional practice was conducted on a credit basis, and in July 1942 he employed the plaintiff attorney to collect his regular and delinquent accounts, paying him a salary plus commission for such services. On July 1, 1943 the plaintiff was inducted into the armed forces. During the plaintiff's absence the defendant employed various persons and a collection agency to handle his collections but none of them were paid a salary; they all worked for a straight commission. The plaintiff was discharged in February 1945 and sought reinstatement with the defendant, who offered the plaintiff a permanent position as sole collector in the same territory in which he had worked before the war but solely on a commission basis. This the plaintiff refused, claiming that he was entitled to be reinstated to a position of "like pay," which, he argued, could be accomplished only by paying him a salary plus a commission.

It must be noted, said the court, that the act exempts the employer from making a reinstatement completely equivalent to the former job if "the employer's circumstances had so changed as to make it impossible or unreasonable to do so." We agree, continued the court, that some very clear reason must be given for the refusal on the part of the employer to give reinstatement to the returning veteran according to the express terms of the act. We also agree that "unreasonable" means more than "inconvenient" or "undesirable." The precise question for decision, continued the court, is this: Was the defendant justified in refusing to reinstate the plaintiff on the basis of compensation which plaintiff demands? In other words, has the defendant sustained the burden which rests on him of proving that "[his] circumstances have so changed" between the time when the plaintiff left the employment of the defendant and when he applied for reinstatement "as to make it impossible or unreasonable" for the defendant to restore the plaintiff to a position of "like pay"?

The testimony shows that in 1942 when the plaintiff entered the defendant's employment, the defendant had on his books more than $32,000 in outstanding accounts, representing 4,115 individual accounts. The corresponding figures for 1943 were $24,640 and 3,213 accounts; for 1944, $9,673 and 1,270 accounts; for 1945, $6,078 and only 707 accounts; and in February 1945, at the time when plaintiff sought reinstatement, $5,819, represented by 731 accounts. These figures stand uncontradicted and indicate a very substantial decline in defendant's professional work while plaintiff was in the service, which, defendant testified, was due very largely to the fact that, during the peak period of the war, he was called on to make an abnormal number of eye examinations for workers in local war plants, and that, as war production and the amount of war plant labor became greatly reduced when the war ended, these examinations fell off correspondingly. During October 1942, November 1942 and April 1943 the defendant expended in monthly commissions approximately $235. During the first six months after plaintiff's discharge, defendant expended an average per month of approximately $110, or less than half of the amount spent in the three months referred to. These figures clearly indicate that there is nothing padded, no misrepresentation, in defendant's position. His business showed a very material falling off, which doubtless justified retrenchment along several lines. Was he justified in adhering to this retrenchment in outlay for collecting accounts when the plaintiff sought restoration of his former position? We believe, said the court, that the defendant was so justified.

To decide otherwise would be tantamount to saying that, although there is no need to have a particular type of position in a given business, nevertheless the act requires that it be set up again for the benefit of the ex-serviceman. Here there is no question of competition or personality involved, but merely the abolition of a salary arrangement, long prior to the return of the plaintiff, because the defendant decided, reasonably and honestly, as we believe, owing to a very substantial reduction in the number of those whose eyes he examined, that he no longer required the services of a salaried collector. The court accordingly entered a judgment in favor of the defendant physician.—*Myers v. Barenburg, 68 F. Supp., 697 (1946).*

Medical Motion Pictures

FILM REVIEWS

Errors of Refraction. Directed by William Alfred Mann, M.S., M.D., and Emile George Nadeau, B.S., M.D. Produced in 1944 by Northwestern University Medical School. Photography by Mervin W. LaRue. 16 mm., color, silent, 1,500 feet (one reel). Showing time fifty minutes. Procurable by rental, $4, or by purchase from Audio-Visual Medical Education Department, Northwestern University Medical School, 303 East Chicago Avenue, Chicago 11.

The purpose of this film is to present the basic principles involved in the optical properties of lenses and the refractive errors of the eye. The subjects included are simple refraction, prisms, convex lenses, various forms of convex lenses, concave lenses, cylindric lenses, the normal eye, the formation of an image, accommodation, presbyopia, emmetropia-myopia hyperopia, myopia, hyperopia and astigmatism.

This film, done entirely in animation, begins with a series of colored perspective pictures showing the deflection of light by passage through glass in the form of rectangular blocks, prisms, cylinders and other solid forms. It shows the structure of the human eye and how an external object, consisting of a pattern of points, becomes represented by an inverted pattern of point images on the retina. Colored diagrams illustrating the meaning of "diopter" serve to introduce a discussion of accommodation, hyperopia, myopia and presbyopia.

The question arises whether the film teaches anything that could not be taught more accurately and at less expense by a book of black and white diagrams and a precise use of mathematical terms. The film labors the simple and obvious and gets into difficulty as soon as it approaches even such simple concepts as that of the conoid of Sturm (misspelled Stürm in the film). More difficult concepts like that of the toric lens, the movements of images in the plane and concave mirrors used in retinoscopy, corresponding points and the horopter are the ones on which medical students need help. It is evident that the difficulty of illustrating these ideas by a movie would be huge.

The film would be useful early in a course in ophthalmology or in physiologic optics as a loose review of elementary facts, to give a glow of color, as it were, to the subject. It is not sufficiently detailed to constitute a good introduction by itself, and since it does not undertake to illustrate anything whatever of an advanced nature it is not likely to prove useful in later work.

Removal of Dermoid Cyst of the Orbit. Prepared in 1942 by Rudolph Jaeger, M.D., Jefferson Hospital, Philadelphia. 16 mm., color, silent, 400 feet (one reel). Showing time sixteen minutes. Procurable on loan from Author, Jefferson Hospital, 10th and Sansom streets, Philadelphia 7.

This short film is interesting and instructive. Most of the photography is fairly good. Occasionally the focus is not changed quickly enough for the manipulations involved. The technic of the operation is good, but there is not much documentation. There is no history of the patient, no differential diagnosis and but little follow-up of the patient. In this type of case ocular movements are of paramount importance and, although the pupils are equal in the film, the patient is not asked to look in all directions, which would demonstrate normal ocular movements. The actual removal of the tumor is not obvious to the onlooker. The cosmetic result is good.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

NATIONAL BOARD OF MEDICAL EXAMINERS EXAMINING BOARDS IN SPECIALTIES

Examinations of the National Board of Medical Examiners and the Examining Boards in Specialties were published in THE JOURNAL, July 19, page 1042.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 22-24, 1948. Sec., Dr. D. G. Gill, 519 Dexter Ave., Montgomery 4.

ARKANSAS: *Eclectic.** Little Rock, Nov. 6. Sec., Dr. Clarence H. Young, 1415 Main St., Little Rock.

CALIFORNIA: *Written.* Los Angeles, Aug. 25-28. *Oral.* San Francisco, Nov. 8-9. Sec., Dr. Frederick N. Scatena, 1020 N. St., Sacramento 14.

DISTRICT OF COLUMBIA: * *Endorsement.* Washington, Sept. 8. Sec., Commission on Licensure, Dr. G. C. Ruhland, 6012 Municipal Bldg., Washington.

FLORIDA: * *Examination.* Jacksonville, Nov. 25-26. Sec., Dr. Harold D. Van Schaick, 6675 Windsor Lane, La Gorce Island, Miami.

GEORGIA: Atlanta, Oct. 14-16. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

ILLINOIS: Chicago, Oct. 14-16. Supt. of Registration, Dept. of Registration and Education, Mr. Philip M. Harman, Springfield.

INDIANA: *Examination.* Indianapolis, June 1948. Sec., Board of Medical Registration and Examination, Dr. Paul R. Tindall, K of P Bldg., Indianapolis.

KANSAS: Topeka, Dec. 3-4. Sec., Board of Medical Registration and Examination, Dr. J. F. Hassig, 905 North 7th St., Kansas City.

KENTUCKY: *Examination.* Louisville, Sept. 22-24. Sec., State Board of Health, Dr. P. E. Blackerby, 620 S. Third St., Louisville 2.

MARYLAND: *Medical Examination.* Baltimore, Dec. 9-12. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore. *Homeopathic Examination.* Baltimore, Dec. 9-10. Sec., Dr. J. A. Evans, 612 W. 40th St., Baltimore.

MINNESOTA: * St. Paul, Sept. 3-5. Sec., Dr. J. F. DuBois, 230 Lowry Medical Arts Bldg., St. Paul.

MISSISSIPPI: *Endorsement.* Jackson, Dec. 1947. Asst. Sec., State Board of Health, Dr. R. N. Whitfield, Jackson.

MONTANA: Helena, Oct. 6-8. Sec., Dr. O. G. Klein, First National Bank Bldg., Helena.

NEVADA: Carson City, Nov. 3. Sec., Dr. G. H. Ross, 215 N. Carson St., Carson City.

NEW HAMPSHIRE: Concord, Sept. 11-12. Sec., Board of Registration in Medicine, Dr. John S. Wheeler, 107 State House, Concord.

NEW JERSEY: *Examination.* Trenton, Oct. 21-22. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO: * Santa Fe, Oct. 13-14. Sec., Dr. LeGrand Ward, 141 E. Palace Ave., Santa Fe.

NORTH CAROLINA: *Endorsement.* Morehead City, July 31. Sec., Dr. Ivan Proctor, 226 Hillsboro St., Raleigh.

RHODE ISLAND: * *Examination.* Providence, Oct. 2-3. Chief, Division of Professional Regulation, 366 State Office Bldg., Providence.

SOUTH CAROLINA: Columbia, Nov. 10-12. Sec., Dr. N. B. Heyward, 1329 Blanding St., Columbia.

WEST VIRGINIA: *Examination.* Charleston, Oct. 2-4. Commissioner, Public Health Council, Dr. N. H. Dyer, State Capitol, Charleston.

WYOMING: *Examination.* Cheyenne, Oct. 6. Sec., Dr. G. M. Anderson, Capitol Bldg., Cheyenne.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: Juneau, First week in August. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

ARIZONA: Sept. 16. Sec., Mr. Francis A. Roy, Science Hall, University of Arizona, Tucson.

COLORADO: *Examination.* Denver, Sept. 17-18. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver.

DISTRICT OF COLUMBIA: *Examination.* Washington, Oct. 20-21. Sec., Commission on Licensure, Dr. G. C. Ruhland, 6012 E. Municipal Bldg., Washington.

FLORIDA: *Examination.* Gainesville, Nov. 1. Final date for filing application is Oct. 16. Sec., Dr. W. M. Emmell, University of Florida, Gainesville.

MICHIGAN: *Examination.* Ann Arbor and Detroit, Oct. 10-11. Sec., Miss Eloise LeBeau, 101 N. Walnut St., Lansing.

MINNESOTA: *Examination.* Minneapolis, Oct. 7-8. Sec., Dr. Raymond N. Bieter, 105 M. Hall, University of Minnesota, Minneapolis.

NEBRASKA: *Examination.* Omaha, Oct. 7-8. Dir., Bureau of Examining Boards, Mr. Oscar F. Humble, State Department of Health, Room 1009, State Capitol Bldg., Lincoln 9.

NEW MEXICO: *Examination.* Santa Fe, Aug. 3. Sec., Miss Marion M. Rhea, % State Land Office, Santa Fe.

OKLAHOMA: Spring 1948. Sec., Dr. Frank Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

OREGON: *Examination.* Portland, Sept. 6. Final date for filing application is Aug. 20. Sec., Board of Higher Education, Basic Science Examining Committee, Mr. C. D. Byrne, University of Oregon, Eugene.

RHODE ISLAND: *Examination.* Aug. 13. Chief, Division of Professional Regulation, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

WISCONSIN: *Examination.* Madison, Sept. 27. Final date for filing application is Sept. 20. Sec., Mr. William H. Barber, Watson and Scott Sts., Ripon.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1937 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied with stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Medical Sciences, Philadelphia

213:385-512 (April) 1947

*Clinical Features of Patent Ductus Arteriosus, with Special Reference to Cardiac Murmurs. S. A. Levine and A. E. Geremia.—p. 385.
*Infectious Hepatitis: Clinical and Laboratory Features of 295 Cases. H. J. Zimmerman, C. F. Lowry, K. Uyeyama and R. Reiser.—p. 395.
Liver Dysfunction in Rheumatic Heart Disease: Preliminary Report. R. W. Kissane, R. S. Fidler and T. E. Clark.—p. 410.
Treatment of Pneumococcic Pneumonia with Oral and Intramuscular Penicillin. H. F. Dowling, Georgine Rotman-Kava, H. H. Hussey and H. L. Hirsh.—p. 413.
*Pharmacologic and Clinical Experiences with Dimethylaminoethyl Benzhydryl Ether Hydrochloride (Benadryl). T. H. McGavack, H. Elias and L. J. Boyd.—p. 418.
Intravenous Use of Human Ascitic Fluid in Shock, Nephrosis and Allied Conditions. R. D. Molina, H. A. Santos and M. M. Alimurung.—p. 435.
Renal Excretory Function and Diet in Diabetes Insipidus. S. B. Beaser.—p. 441.
Observations on Treatment of Carcinoma of Prostate by Orchidectomy. E. K. Landsteiner and H. P. Brown.—p. 450.
Femoral Bone Marrow Cells of the Albino Rat. Mildred Vogel.—p. 456.
Postpartum Blood: Clotting Mechanism and Relationship to Peripheral Blood Picture. A. C. Barnes.—p. 463.
Effect of Circulatory Factors on Bromsulphalein Test in Liver Disease. N. Blumberg and E. M. Schloss.—p. 470.
Menopausal Hypertension: Critical Study. R. D. Taylor, A. C. Corcoran and I. H. Page.—p. 475.
Amylase Levels During Mumps: Findings in Blood and Saliva. I. J. Wolman, B. Evans, S. Lasker and K. Jaegge.—p. 477.
Failure of Massive Salicylate Therapy to Suppress the Inflammatory Reaction in Rheumatic Fever. T. N. Harris.—p. 482.
Changes in Personality Appraisal Associated with Restricted Intake of B Vitamins and Protein. C. R. Henderson, N. C. Wheeler, H. C. Johnson and others.—p. 488.
Virus Pyogen and Virus Pyogen Photosensitivity Relationships in Cutaneous Disease. J. H. Stokes and H. Beerman.—p. 494.
Throat Medication: Survey of Current Trends. N. D. Fabricant.—p. 502.

Patent Ductus Arteriosus and Cardiac Murmurs.—Levine and Geremia studied 37 surgically verified cases of patent ductus arteriosus, with especial attention to the auscultatory signs. Many patients showed a moderate diastolic murmur. The diastolic component was never louder than the systolic; occasionally it was of the same intensity but generally it was fainter. A peculiarity commonly observed was that the murmur appeared to become louder during the latter part of systole, enveloping the second heart sound and continuing with decreasing intensity during diastole. A palpable systolic thrill was present in 21 of 37 cases. This was maximum in the second and occasionally in the first left interspace. It was always systolic in time but occasionally continued well into diastole. In doubtful or puzzling cases catheterization of the heart will be necessary to establish an accurate preoperative diagnosis and avoid harmful surgery.

Clinical and Laboratory Features of Infectious Hepatitis.—Zimmerman and his associates studied 295 cases admitted to a general hospital in eastern France. On the day after entry the blood count, icterus index and urine examinations were performed. Blood icterus determinations and pigment studies of the urine were repeated at three day intervals throughout the hospital stay. Other laboratory studies were performed as indicated. Different disease patterns were differentiated depending on the presence or absence of jaundice, occurrence of relapses and severity. The incubation period was estimated to fall within the accepted three to six week range. Onset was categorized on the basis of the insidiousness or rapidity of the symptom evolution and presence or prominence of systemic and respiratory symptoms. Anorexia, dark urine, malaise, nausea and abdominal pain were the most common symptoms. Jaundice, tender hepatomegaly, adenopathy and occasional splenomegaly were the significant signs. Duration of signs and symptoms was rarely less than five weeks, and many patients had residual disease after eight weeks. Although no deaths occurred, there were 6 cases which showed a clinical picture said to herald a fatal outcome. Most complications were mild. Gingivitis, skin petechiae, melena, epistaxis and "liver spots" occurred. The typical hemogram revealed no anemia but a tendency to reversal of the lymphocyte-neutrophil ratio and slight increase in eosinophils. Elevated serum globulin is of diagnostic and prognostic significance. Patients with deep jaundice, bradycardia and itching had bile salts in the urine. Rest and a diet with high protein and low fat content are the most important factors in treatment.

Pharmacologic and Clinical Experiences with Benadryl.—McGavack and his associates observed 60 normal subjects and 242 patients in order to obtain pharmacologic data and evaluate the therapeutic range and the unpleasant side effects of benadryl. Benadryl is an alkamine ether with a powerful antihistamine action. This is indicated by its ability to suppress completely the dermal and gastric responses to histamine and to decrease capillary permeability. These effects are further demonstrated through the clinical relief afforded patients with diseases in which sudden and/or prolonged releases of excessive quantities of histamine play a pathogenic role. An atropine-like activity for benadryl is suggested by its effects on the eye when applied topically. The variations in blood pressure may simulate one phase of the action of atropine. Disturbances in the sensorium observed in some of the patients receiving the drug have suggested a scopolamine-like action. From the clinical point of view it can be said that benadryl is an exceedingly potent antihistamine and antispasmodic drug which lacks unpleasant cardiovascular and nervous side effects attendant on the use of sympathomimetic agents such as epinephrine, ephedrine and amphetamine. Toxic reactions, while common, rarely preclude its continued use.

American Journal of Medicine, New York

2:419-548 (May) 1947

Symposium on Streptomycin

Streptomycin: General Considerations, Tests for Bacterial Sensitivity and Methods of Measurement of Streptomycin in Body Fluids. W. E. Herrell and F. R. Heilman.—p. 421.
*Streptomycin in Tuberculosis. H. C. Hinshaw, Marjorie M. Pyle and W. H. Feldman.—p. 429.
Use of Streptomycin in Treatment of Bacterial Endocarditis. T. H. Hunter.—p. 436.
Streptomycin in Peritonitis. H. A. Zintel.—p. 443.
Topical Use of Streptomycin in Wounds. E. L. Howes.—p. 449.
Present Status of Treatment for Influenzal Meningitis. Hattie E. Alexander and Grace Leidy.—p. 457.
*Treatment of Tularemia with Streptomycin. L. Foshay.—p. 467.
Treatment of Urinary Tract Infections with Streptomycin. W. L. Hewitt.—p. 474.
Streptomycin Therapy in Undulant Fever. G. H. Finch.—p. 485.
Toxicity of Streptomycin. W. McDermott.—p. 491.

Streptomycin in Tuberculosis.—Hinshaw and his associates administered streptomycin to more than 100 patients with tuberculosis of various types. Experience with this antibiotic agent proved that tuberculosis is amenable to antibacterial therapy. Streptomycin is predominantly bacteriostatic rather than bactericidal, and its therapeutic value is limited by the fact that after exposure to streptomycin for weeks or months strains of Mycobacterium tuberculosis which are resistant to the effects of the drug may be isolated. Hence streptomycin is of most value in conditions in which temporary suppression of the infection will enable the patient to gain the ascendancy over his disease, as in cases of hematogenic tuberculosis, including generalized miliary tuberculosis and tuberculous meningitis. For these conditions large doses (1 to 3 Gm. daily) must be given parenterally for several months. For meningitis intrathecal injections are imperative also during the early weeks of treatment. Exudative pulmonary disease, ulcerating lesions of the respiratory tract and tuberculous draining sinuses have responded to treatment with streptomycin. It has some place in the treatment of bilateral renal tuberculosis or tuberculosis of a solitary kidney. It is also used before and

after thoracic surgery for pulmonary tuberculosis. Because of the potential toxicity, use of the drug may be contraindicated in conditions which will respond satisfactorily to the usual methods of treatment.

Streptomycin in Tularemia.—Foshay employed streptomycin in the treatment of patients of an average age of 36 years with tularemia. One patient died, giving a fatality rate of 2.7 per cent for the series. The time of onset of therapy ranged from the second to the one hundred and twenty-third day of disease, with the average on the twenty-second day. Streptomycin was administered by intramuscular injection every three hours, occasionally every four hours. A few dangerously ill patients received part of the total amount by continuous intravenous or subcutaneous drips. The periods of administration varied from two to seventeen days and the total dosage from 0.64 to 29.5 Gm. A duration of disease of less than two months and a therapy-to-recovery interval of considerably less than forty-six days are new low constants for tularemia. Four patients were desperately ill, exhibiting the highly febrile, incontinent, stuporous, typhoid state. Transitions from this to one of cheerful competence with sustained progressive improvement were effected within seventy-two hours. In 10 patients with pneumonia and pulmonary exudates streptomycin therapy apparently effected a considerable reduction in the resolution time and, on the average, the less the interval between detection of pneumonia and institution of therapy the shorter was the period of repair. Although the experience is too small to permit formulation of an optimal dosage, no evidence has yet appeared that either (1) 0.5 Gm. per day for two days followed by 0.25 Gm. per day for four days or (2) 0.5 Gm. per day for six days is not adequate dosage for the case of usual severity, with or without tularemic pneumonia.

American J. Obstetrics and Gynecology, St. Louis

53:541-720 (April) 1947. **Partial Index**

Fibrous Nature of Human Cervix and Its Relation to Isthmic Segment in Gravid and Nongravid Uteri. D. N. Danforth.—p. 541.
Amniotic Duct as the Key Structure in Determination of the Direction of Growth of Human Placenta and Its Orientation in Uterus. J. Krafka Jr. and L. Bowles.—p. 561.
*Clinical Significance of Rh Antibodies (Rh Agglutinins and Blocking Antibodies) in Serums of Rh-Negative Mothers: Study of 179 Cases. Joan Howard, Marjorie L. Hunt and Barbara C. McIvor.—p. 569.
False Positive Rh Typing Obtained with Commercial Anti-Rh Serum. R. D. Pettit and E. E. Evans.—p. 596.
Effect of Wartime Starvation in Holland on Pregnancy and Its Product. C. A. Smith.—p. 599.
*Selective Cytology Smear for Diagnosis of Cancer. J. E. Ayre.—p. 609.
Operation for Cure of Incontinence of Urine in Female. R. T. Frank. —p. 618.
Irregular Shedding of Endometrium. J. L. McKelvey and L. T. Samuels. —p. 627.
*Time of Ovulation. J. I. Brewer and H. O. Jones.—p. 637.
Placental Metabolism of Vitamin C: I. Normal Placental Content. A. C. Barnes.—p. 645.
Possible Etiologic Significance of Thrombosis of Placental Vein on Mechanism of Placental Infarction and Associated Toxemia of Pregnancy. R. A. Bartholomew.—p. 650.
Podophyllin Treatment of Soft Papillomas of Female Urethra. W. J. Reich, M. J. Nechtow and M. W. Rubenstein.—p. 658.
Uterine Adenomyosis. W. C. Hunter, L. L. Smith and W. C. Reiner. —p. 663.
Metastatic Chorionepithelioma of Lung Treated by Lobectomy. H. C. Maier and H. C. Taylor Jr.—p. 674.
Preliminary Clinical Report on New Synthetic Estrogen Meprane. S. H. Sturgis.—p. 678.
Meningitis Following Continuous Caudal Anesthesia. W. W. Brown Jr: —p. 682.
Dangers of Intracervical Pessary as a Contraceptive Device. S. L. Israel and D. G. Harrel.—p. 684.

Rh Antibodies in Rh-Negative Mothers.—Howard and her associates present an analysis of laboratory observations in 179 pregnant Rh-negative women. The study was undertaken in order to determine the relationships which might exist between the serologic data observed in the mothers and the clinical conditions found in the infants born to them, with the ultimate purpose of presenting the obstetrician with an antepartum interpretation of the significance of the laboratory findings in Rh-negative patients. The data were classified according to the Rh characteristic and the clinical conditions of the infant: group I, normal Rh-negative infants; group II, normal Rh-positive infants; group III, Rh-positive infants with subclinical hemolytic disease of the newborn, and group IV, Rh-positive infants with frank hemolytic disease of the newborn. There were 61 women in group I, 78 in group II, 17 in group III and 20 in group IV. In general, no patient demonstrating a high antepartum titer of either antibody, especially if their presence was detected early in pregnancy, was delivered of a clinically normal Rh-positive infant. A comparison of the occurrence of Rh agglutinin and blocking antibodies indicates that no definite cause and effect relationship between them can be inferred. There appears to be an apparent reciprocity between the titer trends of these two antibodies the significance of which is not entirely clear. The Rh agglutinin appears to be demonstrable in the maternal serum by the twentieth week ante partum. The blocking antibody follows the appearance of the Rh agglutinin and may not be present in demonstrable amounts earlier than about the tenth week antepartum. The hypothesis is suggested that blocking antibody may be produced not as a defense for the mother as is Rh agglutinin but as a protection for the erythrocytes of the fetus.

Selective Cytology Smear for Diagnosis of Cancer.—According to Ayre the original technic for the diagnosis of uterine cancer by the vaginal smear method was arduous and time consuming in the microscopic search for the specifically malignant cells sometimes obscured by the cellular desquamata, leukocytes and blood. Ayre describes a selective cytology technic which was developed in order to obtain evidence of cancer growth in the cells which become earliest involved in cervical cancer. The squamous cells encircling the tiny cervical opening at the squamocolumnar junction constitute a key point of origin for cancer. The spatula test offers a technic providing "surface biopsy" information of these cells prior to their desquamation. By study of these cells, morphologic changes have been identified believed to constitute a precancer cell complex. Many of these so-called precancer cases have shown clinically normal cervices, but the biopsies have verified the suspicious nature of the lesions. The selective cytology technic has been found to be more accurate as an indicator of endogenous estrogen by cornification counts in smears from the cervix and vagina in cancer cases. The previous aspiration method failed to reveal the true cornification level because of excessive amounts of blood and leukocytes obscuring the picture. Through early diagnosis of cancer by routine cytology tests, death from cancer of the cervix should become highly preventable.

Time of Ovulation.—The material comprising this report by Brewer and Jones was obtained from 100 women who were in active menstrual life and who had normal menstrual cycles. The material consists of surgical specimens of the ovary, ovaries or resected portions of ovaries and endometrium. The 100 patients were operated on on various days of the cycle. Days 3 and 27 were the only two days on which no patient was operated on. Diagrams with numbered and unnumbered blocks indicate day of operation, day of ovulation and estimated age of corpus luteum. Fifty-four of the 100 patients had ovulated during the present cycle studied. The range of time of ovulation is from the eighth through the nineteenth day of the cycle. The greatest number ovulated on days 12, 13, 14 and 15, with the highest peak on day 14. The author also reviews observations on ovulation made by other investigators and says that the consistency of the various reports, many of which are based on determination of age of corpora lutea, indicate that it is possible to estimate the age of corpora lutea with reasonable accuracy by histologic and cytologic methods.

American Journal of Public Health, New York

37:253-352 (March) 1947. **Partial Index**

Health Department and Medical Care: Responsibility of Health Officer in Medical Care Program. F. W. Jackson.—p. 253.
Today's Key to Better Public Health Service. W. P. Shepard and R. M. Atwater.—p. 278.
*Universal Serologic Reactivity with Lipid Antigens: Basis for "False Positives." R. L. Kahn.—p. 283.
Colorado Tick Fever. L. Florio and Mabel O. Stewart.—p. 293.
The Nurse's Role in Health Education in Industry. B. J. McGrath. —p. 298.

Universal Serologic Reactivity with Lipid Antigens.—Kahn presents data which show that lipid antigen and serum employed in tests for syphilis can be so treated technically as to elicit positive precipitation reactions in most nonsyphilitic per-

sons. These reactions are referred to as universal serologic reactions. They can be elicited in serum lipid antigen combinations under a variety of technical conditions. Low sodium chloride concentration and low temperature are optimal in eliciting these reactions, and the precipitates formed are generally dispersible in concentrated sodium chloride solution. Low salt concentration and low temperature are also optimal for false positive reactions, and the precipitates formed are generally dispersible in concentrated sodium chloride solution. The fact that the same technical conditions are optimal for universal and false positive reactions suggested a close relationship between these two reactions, and the concept that false positives are highly potent universal reactions which have passed the serodiagnostic threshold and have become manifest with serodiagnostic tests. In agreement with this concept, an illustrative case of a weekly false positive reacting serum is presented in which the universal reaction is of such high potency that it reaches into the serodiagnostic zone and shows positivity in that zone.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

57:409-544 (April) 1947

Electrokymograph for Recording Heart Motion, Improved Type. G. C. Henny, B. R. Boone and W. E. Chamberlain.—p. 409.
*Roentgen Manifestations in Skull of Metastatic Carotid Body Tumor (Paraganglioma), of Meningioma and of Mucocele: Report of 3 Unusual Cases. E. P. Pendergrass and D. Kirsh.—p. 417.
Primary Bronchogenic Cancer Mistakenly Diagnosed as Pulmonary Tuberculosis: Case Reports with Body Section Roentgenography. W. R. Oechsli and K. B. Olson.—p. 429.
*The Vallecular Sign: Diagnosis and Clinical Significance. J. Arendt and A. Wolf.—p. 435.
Congenital Aplasia of Lung: Case Report. W. M. Nesbit, L. W. Paul and W. S. Middleton.—p. 446.
Diagnosis of Ascaris Infestation by Serial Roentgen Examination of Small Intestine. J. E. Lofstrom and D. A. Koch.—p. 449.
Examination of Urinary and Lower Intestinal Tracts Before Treatment of Carcinoma of Cervix Uteri. L. A. Pomeroy.—p. 453.
Congenital Malformations Induced in Rats by Roentgen Rays: Skeletal Changes in Offspring Following a Single Irradiation of the Mother. J. Warkany and Elizabeth Schraffenberger.—p. 455.
Correlation of Tissue Dose and Clinical Response in Irradiation of Bone Tumors and of Normal Bone. Helen Q. Woodard and B. L. Coley.—p. 464.
Prominent Inner Malleolus: Cause of Painful and Enlarged Ankle Region. C. J. Sutro.—p. 472.
Appearance and Growth of Ossification Centers and Increases in the Body Dimensions of White and Negro Infants. Harriet J. Kelly and L. Reynolds.—p. 477.

Manifestations in Skull of Metastatic Carotid Body Tumor, of Meningioma and of Mucocele.—Pendergrass and Kirsh present the clinical, roentgenologic and pathologic findings in 3 patients with osteolytic lesions of the skull. The underlying lesions were a metastatic carotid body tumor, a meningioma and a mucocele. All of these patients had certain similar physical findings, such as exophthalmos. The patient with the carotid body tumor when first seen in another institution had a left Horner's syndrome, a left frontal mass and a mass in the left side of the neck in the region of the left carotid bulb which had been present all his life. A diagnosis of carotid body tumor with metastasis to the frontal area was suggested. He subsequently went to another hospital, where the carotid ligation was performed for the supposed arteriovenous aneurysm. At the time of admission to the University Hospital, the frontal mass was so large that the left eye was completely closed. There was a mass on the left side of the neck, but its significance was not suspected. The roentgenograms revealed an osteolytic lesion of the frontal bone. In the absence of any knowledge of a primary lesion elsewhere one would have to consider meningioma, osteogenic sarcoma and metastatic cancer. The diagnosis in such cases is established only by the correlation of clinical, roentgenologic and pathologic findings. The patient with the meningioma was first believed to have a rather unusual osteolytic type of osteogenic sarcoma. Mucocele is most common in the frontal and ethmoid sinuses. Clinically it is characterized by its long duration, insidious development, lack of insistent symptoms and distention of cavities. Roentgenographically one observes enlargement of the sinus with pressure erosion of the walls, resulting in increased radiolucency. The periphery of the bone defect is often quite dense, as the result of new bone formation. If the mucocele remains untreated, large areas of bone destruction occur and the lesion encroaches on adjacent structures and even causes exophthalmos.

The Vallecular Sign.—Arendt and Wolf assert that the dysphagia which accompanies many malignant growths of the hypopharynx of the esophagus and the upper stomach region is not due to stenosis and prestenotic regurgitation but is primarily a disturbance of deglutition. The symptom of barium retention in the valleculae and in the pyriform sinuses is found in many of these cases. The presence of this vallecular sign is in favor of an organic lesion and is against a functional type of dysphagia. A positive vallecular sign is demonstrated in carcinoma of the cardia, of the hypopharynx and the esophagus, also in bulbar paralysis and in myasthenia gravis. The occurrence of the vallecular sign in myasthenia gravis and bulbar paralysis is helpful in the recognition of these diseases, and its improvement is a good measure of the therapeutic effect of thymus irradiation.

American Journal of Surgery, New York

73:529-634 (May) 1947

Postoperative Pulmonary Atelectasis. A. L. Kruger, P. S. Marcus and M. T. Hoerner.—p. 531.
Caudal Transsacral Anesthesia in Rectal Surgery: Routine Use in More Than 2,000 Cases. C. Eaton.—p. 540.
Injuries to Semilunar Cartilages of Knee Joint. Marion K. King and W. S. Hotchkiss.—p. 544.
Maxillofacial Injuries: Reconstructive Surgery of Dehiscent Parotid Duct and Dehiscent Peripheral Facial Nerve. R. C. Seeley.—p. 551.
Late Repair of Massive Tissue Defects by Split Skin-Lined Flap Graft. W. B. Macomber and L. R. Rubin.—p. 564.
Surgical Approach to Tibial Nerve Below Popliteal Fossa. A. M. DeAngelis.—p. 568.
Continuous, Vertical Mattress Suture: Application and Usefulness. J. A. Davis.—p. 576.
Experiences with Pulsating Hematoma. W. C. Beck.—p. 580.
Method for Repair of Posterior Tibial Nerve. F. F. Allbritten Jr.—p. 588.
Simple Technic of Suturing in Gastrointestinal Anastomoses. H. Sneierson.—p. 592.
Reduction of Rectal Prolapse. J. F. Wenzel.—p. 596.

American Review of Tuberculosis, New York

55:195-296 (March) 1947

American Association for Thoracic Surgery: Reports of Its Meeting on May 29, 30 and 31, 1946. R. H. Meade Jr.—p. 195.
*Pulmonary Resection in Treatment of Pulmonary Tuberculosis: Analysis of 88 Patients Followed for Period of Two to Twelve Years After Operation. R. H. Overholt, N. J. Wilson, J. T. Szypulski and L. Langer.—p. 198.
Oleothorax. J. H. Moyer Jr.—p. 223.
*BCG Vaccination in Scandinavia: Twenty Years of Uninterrupted Vaccination Against Tuberculosis. K. Birkhaug.—p. 234.
Control of Tuberculosis in Brazil: New Orientation—A New Mentality. M. M. Bueno.—p. 250.
Intravenous Infection of Chick Embryo with Tubercle Bacilli: Inhibitory Effects of Streptomycin. H. F. Lee and A. B. Stavitsky.—p. 262.
*Effect of Streptomycin on Tubercle Bacillus: Use of Dubos and Other Mediums in Tests for Streptomycin Sensitivity. E. Wolinsky and W. Steenken Jr.—p. 281.

Pulmonary Resection in Pulmonary Tuberculosis.—Overholt and his associates analyze a group of 88 patients treated by 92 resections between 1934 and March 31, 1944. The follow-up period ranged from two to twelve years. March 31, 1944 was chosen as the dividing line for two reasons. A minimum follow-up period of two years was made possible and this date ushered in an era of new technics. Since then, general anesthesia and the side position of the patient during operation have been abandoned. During the past two years local anesthesia and the face down position have been used. Improved operative technic has reduced the serious complications following resection. Ipsilateral exacerbation of remaining tuberculous foci has been the most common cause of failure. The role of postlobectomy thoracoplasty in its prevention has been emphasized. The operative fatality at present for both lobectomy and pneumonectomy is approximately 5 per cent for patients who are reasonable risks. In the desperate risk group the operative fatality is 43.8 per cent. Of the patients followed from two to twelve years 42.4 per cent of the lobectomy series and 48.1 per cent of the pneumonectomy series are clinically well with negative sputum; 18.2 per cent of those treated with lobectomy and 41.2 per cent of those treated with pneumonectomy are dead.

Twenty Years of BCG Vaccination in Scandinavia.—Birkhaug points out that during the past forty years a remarkable shift has been observed in Scandinavia in the age period when the primary tuberculous infection takes place. At the turn of the century about 50 per cent of persons were tuberculin reactors between 10 and 15 years of age and almost 90 per cent between 25 and 30 years. In 1944 only 20 per cent of the former and 50 per cent of the latter age groups had had contact with tubercle bacilli, and the peak of tuberculous disease was observed in the 20 to 30 aged group. Scandinavian clinicians have observed that the acute and rapidly fatal exudative type of tuberculosis has become common among adolescents and young adults who did not react to tuberculin. They believe that the best hope for controlling this advanced shift in the time of primary tuberculous infection lies in BCG vaccination. The basis for this belief is (*a*) the absolute harmlessness of the BCG vaccine both in animals and in man and (*b*) its ability to produce a mild tuberculin reactivity and a relatively high protection against an exogenous infection with virulent tubercle bacilli. Intracutaneous or transcutaneous BCG vaccination renders nontuberculin reactors tuberculin positive in more than 90 per cent within two months after vaccination. During the period of BCG vaccination allergy and exposure to known tuberculous infection, BCG affords an almost complete protection against the morbid postprimary tuberculous lesions and a relatively high protection against secondary tuberculosis, including clinical pulmonary disease. The basis for the concerted Scandinavian mass BCG vaccination is the importance tuberculosis plays even today as an endemic disease—the commonest chronic disease in persons between 15 and 30 years of age. Since 1940 more than 100,000 persons have been vaccinated with BCG in Norway, 58,000 army conscripts and 250,000 civilians in Sweden and 150,000 persons in Denmark.

Effect of Streptomycin on Tubercle Bacilli.—Three different methods were used by Wolinsky and Steenken for their tests on the effect of streptomycin on the growth of the human type of tubercle bacillus H 37 Rv, namely (1) surface growth on synthetic medium, (2) deep-seated growth in synthetic medium, (3) Dubos's liquid medium. The method utilizing the new liquid medium of Dubos was found to be the most satisfactory. Streptomycin in concentration of 0.05 microgram per cubic centimeter was found to inhibit growth of H 37 Rv slightly, 0.2 microgram per cubic centimeter to inhibit growth definitely and 0.4 microgram per cubic centimeter to inhibit growth completely, with an incubation time of sixteen to twenty days in mediums at p_H 7.1 to 7.4.

Annals of Internal Medicine, Lancaster, Pa.

26:489-642 (April) 1947

Physicians of the Machine Age. D. W. Bronk.—p. 489.
*Rheumatic Pneumonia. D. W. Seldin, H. S. Kaplan and H. Bunting. —p. 496.
Bronchial Spasm in Cardiac Asthma. M. Plotz.—p. 521.
Use of Methyl-Iso-Octenylamine in Migraine. P. S. MacNeal and D. Davis.—p. 526.
*Brucella Sensitization: Clinical Evaluation. W. Darley and R. W. Gordon.—p. 528.
Hepatic Cirrhosis as Complication of Chronic Ulcerative Colitis. H. J. Tumen, J. F. Monaghan and E. Jobb.—p. 542.
Cardiologic Criteria for Diagnosis of Rheumatic Heart Disease in Apparently Healthy Subject. T. N. Harris.—p. 554.
Action of Carbon Dioxide in Water Mobilization. E. C. Mason.—p. 561.
Prescription of Physical Medicine by the Internist. A. L. Watkins. —p. 569.
Bronchopulmonary Actinomycosis. E. B. Kay.—p. 581.

Rheumatic Pneumonia.—According to Seldin and his associates the relationship of rheumatic pneumonia to rheumatic fever is still obscure. The authors report 6 cases of active rheumatic fever with carditis, presenting the characteristic pathologic features of rheumatic pneumonia. A uniform clinical picture was encountered, characterized by an abrupt onset of profound respiratory distress in the absence of commensurate physical signs in the chest. Hacking cough with scanty, blood streaked sputum, moderate to high fever and leukocytosis were also present. The disease was manifested roentgenologically by a bilateral, multilobar, nonsegmental infiltration which resembles pulmonary edema. The clinical and roentgenologic features of rheumatic pneumonia were observed in five of one hundred attacks of active rheumatic fever occurring in 91 patients. No instance of lobar or primary atypical pneumonia was encountered in this series, and pneumonitis secondary to bronchiectasis was found only twice. Rheumatic pneumonias therefore appear to be more frequent than intercurrent pneumonias during the course of active rheumatic fever. All cases of rheumatic pneumonia terminated fatally. This manifestation of rheumatic fever would appear to carry a grave prognosis.

Brucella Sensitization.—Darley and Gordon performed intradermal tests with Huddleson's brucellergen on two groups. The first group consisted of 1,497 adult patients, all having presented themselves for examination because of some health problem. Of these 1,497 patients 236, or 15.76 per cent, were found Brucella sensitive. The second or control group was made up of 1,170 well persons. One hundred and twenty, or 10.25 per cent, gave positive tests. Thus Brucella sensitization occurred significantly more often in the chronically ill than in the well. This would seem to indicate that Brucella infection is frequently involved in chronic ill health and also that sensitization must to a considerable degree parallel active infection. The data obtained were opposed to any idea that Brucella infection might be of etiologic or contributory importance in allergy, chronic arthritis or psychoneurosis. With regard to the question that a low grade indolent form of brucellosis could be identified as a clinical entity, careful clinical study of the Brucella sensitive patient can with reasonable accuracy establish the presence or absence of active infection. Every possible explanation for symptoms must be carefully considered before a diagnosis of chronic brucellosis is justified. Because of the superficial resemblance between psychoneurosis and what is considered chronic brucellosis, the former possibility should always be ruled out. The authors present data on 74 patients in whom a diagnosis of chronic brucellosis was probable. The most frequent symptoms encountered were fatigue, muscle and joint aches and pains, nonmigrainous headache, digestive complaints and low grade fever. The incidence of positive agglutination reactions was high in the group.

Archives of Dermatology and Syphilology, Chicago

55:433-600 (April) 1947

Lichen Sclerosus et Atrophicus with Bullous Lesions and Extensive Involvement. Helen Reller Gottschalk and Zola K. Cooper.—p. 433.
Experiences in Military Dermatology: Interpretation in Plans for Improved General Medical Care. D. M. Pillsbury and C. S. Livingood.—p. 441.
Moeller's Glossitis: Case with Remission During Pregnancy. H. Rattner. —p. 463.
Tuberculoid Leprosy of Lupoid Appearance. O. G. Costa and J. Mariano. —p. 474.
Reticulum, or Lattice, Fibers: Further Studies. S. C. Way.—p. 478.
Role of Food Allergy in Eczematoid Dermatitis. J. M. Flood and D. J. Perry.—p. 493.
Basal Squamous Cell Epithelioma Associated with Leukemia. F. Reiss and W. Konheim.—p. 507.
*Trichophytin in Thromboangiitis Obliterans. J. C. Holman Jr.—p. 512.
Cardiolipin-Lecithin Antigen: Development Toward Single Standard Test of Blood for Syphilis. B. S. Kline.—p. 514.
*Sulfonamide Psoriasiform Dermatitis. O. S. Philpott.—p. 525.
Atypical Lichen Planus Tropicalis. M. G. Butler.—p. 535.

Trichophytin in Thromboangiitis Obliterans.—The use of trichophytin in the treatment of thromboangiitis obliterans or migratory phlebitis has not been carried on to any extent. During the last year Holman saw 2 patients with thromboangiitis obliterans whose response following the use of trichophytin was so dramatic that he feels that some attention should be given to the possibility of the use of trichophytin as a therapeutic agent in this disease. The first patient, who had had thromboangiitis obliterans for years, was given a trichophytin test, which resulted in an erythematous, edematous and urticarial reaction which persisted so that the patient became mentally upset and had to be hospitalized for about three weeks. The urticaria was controlled and the feet were for the first time warm and comfortable. When the thromboangiitis obliterans recurred two months later, Holman gave the patient trichophytin in a dilution of 1:240 once a week for four weeks and 1:120 once a week for four weeks. He was relieved of all symptoms of thromboangiitis obliterans after the second week of treatment and has remained so. The second patient showed a similar favorable response.

Sulfonamide Psoriasiform Dermatitis.—Philpott observed a cutaneous eruption which he thinks is due to sulfonamides. It is a type of psoriasiform dermatitis exhibiting many of the essential features of psoriasis. He reports 3 cases which illustrate some of the clinical features of this condition. All 3 patients were women. In each the eruption followed relatively soon after she had taken a sulfonamide drug; in each the appearance of the eruption warranted a clinical diagnosis of psoriasis, and in 2 of the 3 the microscopic diagnosis was consistent with that of psoriasis. None of the 3 had a suggestive hereditary history; none had previous disturbances of the skin. The duration of the eruption in 1 was four months, and the skin has been clear six months. In another the lesions were present four months and cleared from the body but remained in the scalp for fifteen months and then reappeared and have been present ever since. The third still has lesions and has had them continuously for twenty-one months. This experience points to infective processes as an inciting factor of initial attacks of psoriasis; apparently the sulfonamide compounds may act similarly. Holman suggests the possibility of the existence of psoriatic patients in whom the disease remains below the threshold of the eruptive phase until some potent excitant such as a sulfonamide drug precipitates an eruptive reaction.

Archives of Neurology and Psychiatry, Chicago

57:397-520 (April) 1947

*Cerebral Anoxia from High Altitude Asphyxiation: Clinicopathologic Study of 2 Fatal Cases with Unusually Long Survival and a Clinical Report of a Nonfatal Case. L. A. Titrud and W. Haymaker.—p. 397.
Cerebellar Medulloblastoma in Adults. E. B. Spitz, H. A. Shenkin and F. C. Grant.—p. 417.
Static Tremor with Hemiplegia; Report of a Case: Development, Progression for Seven Years and Postmortem Histologic Observations. F. A. Mettler, L. M. Davidoff and R. Grimes.—p. 423.
*Japanese B Encephalitis: Clinical Observations in Outbreak on Okinawa Shima. L. Lewis, H. G. Taylor, M. B. Sorem and others.—p. 430.
Incidence of Anisocoria and Difference in Size of Palpebral Fissures in 500 Normal Subjects. B. C. Meyer.—p. 464.
Relation of Mental Imagery to Hallucinations. P. F. Durham Seitz and H. B. Molholm.—p. 469.
*Medulloblastoma of Cerebellum, with Survival for Seventeen Years. W. Penfield and W. Feindel.—p. 481.

Cerebral Anoxia from High Altitude Asphyxiation.—Titrud and Haymaker report 3 cases of anoxic anoxia occurring in aviators while engaged in combat. In 2 cases the anoxia proved fatal in forty hours and three weeks, respectively, and the third patient was observed for a period of three weeks and then was transferred. In the first, the accident occurred at an elevation of "more than 20,000 feet," the patient being found unconscious five minutes after completion of a bomb run, at which time resuscitation was commenced. The second patient was exposed to an atmosphere at 24,000 feet for approximately ten minutes. In the third, it was at 27,000 feet that the oxygen tank of the plane was shot away, necessitating a rapid descent to 7,000 feet, during which time no effort could be made to revive the patient. In the case of forty hours' duration necrosis of ganglion cells was observed in the third to sixth laminas of the cerebral cortex, the striatum, the cerebellum, Sommer's sector of the hippocampus and the anterior horns of the spinal cord, mostly focal in character, and degeneration of myelin in the internal capsule. Also observed were early proliferation of the fixed tissue cells of the leptomeninges and vascular adventitia and mild exudation of lymphocytes and neutrophils into the meshes of the arachnoid, the perivascular spaces and, to a limited extent, the parenchyma of the brain. In the case of three weeks' duration the changes were of virtually the same location except that the cerebral white matter, brain stem and spinal cord were relatively spared. The damage in these cases of sudden and severe anoxic anoxia is strikingly similar to that observed in some of the other forms of anoxia.

Japanese B Encephalitis.—Lewis and his associates review the clinical characteristics of Japanese B encephalitis as observed in an outbreak in the civilian population of Okinawa Shima. They present an analysis of symptoms and physical signs in 65 patients and reports of 7 cases illustrating variations in the course and outcome of the disease. The disease was most common among children, resulting in a mortality of 20 per cent, and caused permanent disorder of the nervous system in at least one fifth of the patients. The manifestations were characteristic of a diffuse encephalomyelitis and resembled those of other neurotropic virus infections. The gross and microscopic lesions of the nervous system are compared with those of other virus encephalitides.

Medulloblastoma of Cerebellum, with Survival for Seventeen Years.—According to Penfield and Feindel, medulloblastoma of the cerebellum seems invariably fatal in spite of the fact that it is temporarily sensitive to roentgen therapy. Reports in the literature have pointed out the tendency to longer survival from this tumor in adults. In the case reported a medulloblastoma of the cerebellum made its appearance when the patient was 22 years of age. There was recurrence of symptoms after nine years and again after sixteen years. In the first (and probably the second) instance the recurrence was at a distance from the cerebellum, and in each the result of roentgen therapy was initially satisfactory. Although the histologic appearance suggested a high degree of malignancy, there was survival for seventeen years.

Arkansas Medical Society Journal, Fort Smith

43:257-284 (April) 1947

Tuberculosis Control Activities of the Arkansas State Board of Health. A. C. Cuftis.—p. 257.
Consideration of Surgical Complications of Amebiasis of Colon and Rectum. D. B. Stough.—p. 261.
Trends in Tuberculosis Control Procedures. H. R. Edwards.—p. 270.

43:285-310 (May) 1947

Pituitary Ovarian Relationship. J. H. Sanderlin and W. B. Harrell. —p. 285.
Rheumatic Fever in Childhood. S. Phillips.—p. 287.
Diagnosis of Acute Abdomen. G. E. Cannon.—p. 289.

Bull. of U. S. Army Med. Dept., Washington, D. C.

7:413-492 (May) 1947

Carcinoma of the Skin and Lip. E. B. Helwig and R. G. Brown. —p. 437.
Functional Urinary Frequency in the Soldier. K. B. Conger.—p. 441.
Neuropsychiatric Casualties in an Infantry Division. H. D. Nesmith. —p. 445.
*Treatment of Plantar Corns. J. R. Vasko.—p. 455.
Ventilation Rates of Sealed Aircraft Cabins. K. E. Penrod.—p. 460.
Coccidioidal Meningitis. C. B. Whims.—p. 466.

Plantar Corns.—Vasko says that plantar corns are frequently misdiagnosed as plantar calluses and sometimes as plantar warts. Plantar calluses and plantar corns may look alike, but the former are not tender while the corns are exquisitely so. The surest way to discover whether the callus conceals a corn or not is to pare off the top. Should a corn be present, one will encounter a small hard kernel like a rice body. A plantar wart is usually not surrounded by callus. Also a wart has the appearance of the cross section of a nerve and is usually moist and soft, whereas the corn is dry and hard. In the treatment advocated by Vasko the corns are trimmed of excess callus and the hard kernel in the center removed with a small, blunt, round ended knife. The skin underlying this kernel was always pink and rather tender. A subcutaneous injection of 1 cc. of 1 per cent procaine was then made into the base, and it was noticed that considerable pressure had to be used to force the procaine in. Apparently the skin on which the base of the corn rested was adherent to the fibrous element of the subcutaneous fat. The injection was quite uncomfortable for a moment, but after the ballooning of the tissue was accomplished the pain ceased. Painless walking was possible almost at once. In a few cases the relief was permanent, but in the majority some discomfort returned within a few days. The latter group received additional injections. To avoid disappointment in the treatment of verrucae and simple calluses, it is recommended that this treatment be used only on plantar corns.

Delaware State Medical Journal, Wilmington

19:41-58 (March) 1947

Diabetes Mellitus and Gallbladder Disease. E. M. J. Bohan.—p. 41.
Mesenteric Fibrosarcoma: Report of a Case. P. Alva and L. Vazquez. —p. 45.
Metastatic Carcinoma of the Lung Simulating Heart Failure. L. Vazquez.—p. 48.
Successful Immunization Procedures. G. J. Boines.—p. 50.

Endocrinology, Springfield, Ill.

40:231-298 (April) 1947

Interpretation of Estrogenic Assays. J. M. Curtis, J. J. Umberger and Lila F. Knudsen.—p. 231.
Imposition of Diurnal Ovulatory and Temperature Rhythms by Periodic Feeding of Hens Maintained Under Continuous Light. R. M. Fraps, B. H. Neher and I. Rothchild.—p. 241.
Effect of Pregnancy Complicated by Alloxan Diabetes on Fetuses of Dogs, Rabbits and Rats. H. C. Miller.—p. 251.
Effect of Thyroidectomy on the Conversion of Carotene to Vitamin A. V. A. Drill and A. P. Truant.—p. 259.
Regulation of Pituitary Adrenocorticotropic Activity During Response of Rat to Acute Stress. G. Sayers and Marion A. Sayers.—p. 265.
Role of Adrenal Cortex in Mammary Development and Its Relation to Mammogenic Action of Anterior Pituitary. A. T. Cowie and S. J. Folley.—p. 274.
Action in Vitro of Thyrotropic Hormone and Iodide on Thyroid Cells. L. C. Junqueira.—p. 286.

Gastroenterology, Baltimore

8:397-564 (April) 1947

*Giant Hypertrophic Gastritis. S. N. Maimon, J. P. Bartlett, Eleanor M. Humphreys and W. L. Palmer.—p. 397.
Effect of Vagotomy on Gastric Function. S. Wolf and W. DeWitt Andrus.—p. 429.
Rate of Healing of Gastric Ulcers. H. M. Pollard, W. H. Bachrach and M. Block.—p. 435.
Syndrome of Nontropical Sprue with Hitherto Undescribed Lesions of the Intestine. J. Schein.—p. 438.
Oral Cholecystography with Priodax: Correlation with Pathologic Findings. J. L. Jarvis and D. Cayer.—p. 461.
Preliminary Report on Protein Hydrolysate Therapy for Peptic Ulcer. S. T. Kimble Jr.—p. 467.
*Protein Hydrolysate Therapy for Peptic Ulcer: Report of 26 Cases. H. H. Hodges.—p. 476.
Nonreactive Aluminum Hydroxide in Treatment of Peptic Ulcer. F. H. Smith.—p. 494.
Complications Following War Wounds of Abdomen. M. A. McIver.—p. 504.
Treatment of War Wounds Involving Gastrointestinal Tract. R. M. Zollinger.—p. 512.
Primary Megacolon. J. M. Swarts.—p. 519.
Simplified Procedure for Determination of Pepsin and Trypsin Concentrations in Digestive Juices. M. H. F. Friedman.—p. 526.

Giant Hypertrophic Gastritis.—Six cases of giant hypertrophic gastritis, 2 with superimposed hyperplastic polyp formation, are described by Maimon and his co-workers. The difficulty in differentiating this condition from neoplasm is evident from the fact that all 6 were subjected to operation. The symptomatology is indefinite. Anatomically, roentgenologically and gastroscopically the resemblance of the gastric folds to the convolutions of the brain is striking and is perhaps the most salient feature. Microscopic examination reveals redundancy of the mucosa and hypertrophy of the glandular structures, with cyst formations and metaplasia to the intestinal type of epithelium. The pepsinogen and acid forming cells are replaced by the more primitive type. Fibroplasia with increase of connective tissue and edema were prominent features. In 2 cases redundancy was outstanding, while in 2 others hyperplasia and increased thickness of the mucosa were remarkable. Multiple polyps developed in 2 cases.

Protein Hydrolysate Therapy for Peptic Ulcer.—Hodges evaluates the effectiveness of the oral administration of a protein hydrolysate in the treatment of chronic peptic ulcer and makes observations pertaining to nutrition and metabolism in human subjects whose sole source of dietary nitrogen was predigested protein. Twenty-six patients have been selected for this study. Twenty-four had received adequate conventional dietary therapy previously without significant improvement or had suffered a recurrence of symptoms while adhering to such a program. The patients were treated with a protein hydrolysate-carbohydrate mixture for from two to three weeks. In the majority this treatment appeared more efficacious than a conventional dietary program in producing a remission, but the frequency of relapses was not diminished. Three patients failed to respond satisfactorily, one of these suffering a serious gastrointestinal hemorrhage in the midst of therapy. A gain in weight was the rule. A rise in blood urea nitrogen was seen in most of the patients, but the serum protein and hemoglobin changes were inconsistent. The degree of gastric acidity after treatment tended to be reduced. Individual feedings of this mixture completely neutralized free gastric acid for an average of seventy minutes. The beneficial effects of the protein hydrolysate are ascribed to its buffering action and to the high nitrogen intake. It is believed that the protein hydrolysate may become a useful adjunct to the medical management of peptic ulcer but will not prove to be a panacea.

Hawaii Medical Journal, Honolulu

6:221-296 (March-April) 1947

Pulmonary Resection for Tuberculosis. P. W. Gebauer.—p. 233.
Bronchoscopic Diagnosis in Nontuberculosis Chest Conditions. D. B. Radner—p. 236.
Immunologic Factor Involved in Case of Hemolytic Disease of Newborn. L. Welty.—p. 239.
Congenital Heart Disease. F. B. Schultz.—p. 241.

Journal of Bacteriology, Baltimore

53:381-508 (April) 1947. **Partial Index**

Comparative Action of Bromine and Iodine on Toxic Enzymes of Staphylococcus Aureus and Streptococcus Pyogenes. H. Farkas.—p. 401.
Relationship Between Lactobacillus Acidophilus (Moro) Holland and Lactobacillus Casei (Orla-Jensen) Holland. C. S. Pederson.—p. 407.
Growth of Clostridium Septicum and Its Inhibition. F. J. Ryan, Lillian K. Schneider and R. Ballentine.—p. 417.
Nutrition of Protozoa: Simplified Medium for Investigation of Unknown Factors in Blood Serum Essential for the Sustained Growth of Trichomonas Vaginalis. H. Sprince and A. B. Kupferberg.—p. 435.
Effect of Metabolites of Escherichia Coli on Growth of Coli-Aerogenes Bacteria. J. M. Coblentz and M. Levine.—p. 455.
Mechanism for Development of Resistance to Streptomycin and Penicillin. M. Klein.—p. 463.
Antagonism by Aerobacter Strains. E. S. Wynne.—p. 469.
Rapid Method for Staining of Rickettsia Orientalis. F. H. Diercks and R. O. Tibbs.—p. 479.
*Observations on In Vivo and In Vitro Development of Bacterial Resistance to Streptomycin. C. W. Price, W. A. Randall, V. L. Chandler and R. J. Reedy.—p. 481.
Studies on Hemolytic Streptococci: Differentiation of Species in Streptococci of Group A. Alice C. Evans.—p. 489.

Bacterial Resistance to Streptomycin.—Experiments made by Price and his associates revealed that different species of bacteria and different strains of the same species vary greatly in their in vitro resistance to streptomycin and streptothricin. Resistance of organisms to streptomycin was more readily induced than to streptothricin, and to a greater degree. Streptomycin-induced resistance of Eberthella typhosa is accompanied with changes in morphology. In order to make comparative studies of the sensitivities of organisms to streptomycin it is imperative to employ a broth of uniform hydrogen ion concentration containing the same constituents. Furthermore, it is imperative that in testing an organism for sensitivity to streptomycin it be tested in the same batch of broth in which its original sensitivity was determined. It would be well for laboratories to maintain a reference standard medium against which all subsequent batches of medium could be tested with an organism of previously established sensitivity. When rats are fed on a diet containing streptomycin there appear in a relatively short time in the feces of these animals large numbers of microorganisms which are extremely resistant to this antibiotic.

Journal of Clin. Endocrinology, Springfield, Ill.

7:1-78 (Jan.) 1947

Gonadotropic Hormone: Comparison of Ultrafiltration and Alcohol Precipitation Methods of Recovery from Urine. E. C. Jungck, W. O. Maddock and C. G. Heller.—p. 1.
Three Unusual Endocrinopathies with Associated Ovarian Pathology: I. Ovarian Agenesis. II. Precocious Puberty. III. Virilism. Minnie B. Goldberg, Alice F. Maxwell and Pearl M. Smith.—p. 11.
*Paroxysmal Hypertension with Concomitant Swelling of Thyroid Due to Pheochromocytoma of Right Adrenal Gland: Cure by Surgical Removal of Pheochromocytoma. J. Bauer and E. Belt.—p. 30.
Effect of Thiouracil Derivatives on Fetuses and Infants. E. Freiesleben and K. Kjerulf-Jensen.—p. 47.
Thyroid and Adrenal Interrelations, with Special Reference to Hypotrichosis Axillaris in Thyrotoxicosis. R. H. Williams.—p. 52.

Paroxysmal Hypertension.—Bauer and Belt report paroxysmal diffuse swelling of the thyroid accompanying and simultaneous with paroxysmal attacks of hypertension in a woman aged 47. The sudden swelling of the thyroid gland added to the usual signs and symptoms characteristic of the adrenal sympathetic syndrome reached its peak and faded away concurrently with the rise and fall of blood pressure in each hypertensive attack. There were extremely rapid and excessive variations in blood pressure, which usually are not associated with the syn-

drome. A tentative diagnosis of chromaffin tumor was made and corroborated by the finding that paroxysmal hyperglycemia accompanied the attacks. The diagnosis was established when the supposed tumor was visualized in the x-ray plate by perirenal insufflation. A cure was affected by the surgical removal of the tumor under spinal anesthesia. Microscopic examination of the specimen revealed a cystic pheochromocytoma of the adrenal gland.

Journal of Experimental Medicine, New York

85:329-440 (April) 1947. **Partial Index**

*Mode of Action of Streptomycin on Type b Hemophilus Influenzae. Hattie E. Alexander and Grace Leidy.—p. 329.
Normal Structure and Degenerative Changes of Cytoplasm of Liver Cells and Tumor Cells Derived from Them. E. L. Opie.—p. 339.
Effect of Ultraviolet Irradiation on Various Properties of Influenza Viruses. W. Henle and Gertrude Henle.—p. 347.
Impairment of Antibody Response in Pyridoxine Deficient Rats. H. C. Stoerk, H. N. Eisen and Hedda M. John.—p. 365.
Studies on Glycogen Nephrosis in Alloxan Treated Diabetic Rats. G. W. Curtis, S. L. Robbins and I. Glickman.—p. 373.
Manometric Method for Measuring Activity of Clostridium Welchi Lecithinase and Description of Certain Properties of This Enzyme. P. C. Zamecnik, Lydia E. Brewster and F. Lipmann.—p. 381.
Epidemic Diarrheal Disease of Suckling Mice: I. Manifestations, Epidemiology and Attempts to Transmit Disease. F. S. Cheever and J. H. Mueller.—p. 405.
Relation of Size of Inoculum and Age of Infection to the Curative Dose of Penicillin in Experimental Syphilis, with Particular Reference to Feasibility of Its Prophylactic Use. H. Eagle, H. J. Magnuson and R. Fleischman.—p. 423.

Action of Streptomycin on Type b Hemophilus Influenzae.—Alexander and Leidy found that of 14 patients treated with streptomycin alone 10 recovered promptly; in 3 of the 4 remaining cases failure was proved to be due to emergence of resistance of the organisms to streptomycin. The rapid development of resistance to 1,000 units of streptomycin per cubic centimeter during treatment argues against metabolic adaptation of the bacteria. Study of large samples of initial bacterial populations shows resistant members in all ten strains examined. The proportion of resistant members is not significantly higher before treatment in patients who do poorly than in those who respond promptly. The results indicate that emergence of resistance is the result of a selective process; after elimination of sensitive members the very few resistant ones, apparently present in all strains, make up most or all of the population in the cultures taken after commencement of treatment. The survival of organisms which can grow in high concentrations of streptomycin, either in patients or in vitro, is influenced more by the size of the bacterial population than by any other known factor. Since the bacterial population is relatively small in patients with mild or moderately severe infections, the likelihood of a significant number of highly resistant members being present is remote.

Journal of Immunology, Baltimore

55:309-412 (April) 1947

Studies on Botulinum Toxoids, Types A and B: III. Immunization of Man. H. R. Reames, P. J. Kadull, R. D. Housewright and J. B. Wilson.—p. 309.
Quantitative Estimation of Egg and Chicken Proteins in Equine Encephalomyelitis Vaccines. L. L. Engel and R. Randall.—p. 325.
Relations Between Equine Encephalomyelitis Virus (Eastern Type) and Normal Host Cellular Constituents. L. L. Engel and R. Randall.—p. 331.
Quantitative Study of Arthus Phenomenon Induced Passively in the Rabbit. E. E. Fischel and E. A. Kabat.—p. 337.
Effect of Metabolites, Metabolite Antagonists and Enzyme Inhibitors on Growth of Vaccinia Virus in Maitland Type of Tissue Cultures. R. L. Thompson.—p. 345.
*Complement Fixing Antibodies of Lymphogranuloma Venereum in Mice: Their Development and Response to Sulfonamide Therapy. M. J. Wall.—p. 353.
Dysentery: A Progress Report for the Years 1942 to 1946. A. J. Weil.—p. 363.

Antibodies of Lymphogranuloma Venereum.—Wall studied the development of complement fixing antibodies in mice following infection with the virus of lymphogranuloma venereum. Antibody was always demonstrable at the end of two weeks after inoculation. A peak in titer was reached three to five weeks after infection. A high titer persisted throughout the eighteen week period of observation. Virus could be recovered from the brains of the animals at any time after the appearance of signs of illness and throughout the period of recovery. The sulfonamides are effective in the treatment of lymphogranuloma venereum, but animals treated with sulfonamides from the time of inoculation for a period of several weeks gave negative tests for antibodies at the end of this time. In mice treated after the infection had been established, titers of antibody were lower than in control animals and a relationship was found between the end titer and the duration of therapy. When therapy was continued for several weeks, the antibodies disappeared completely. Virus could still be recovered from the tissues of these animals, but the ease with which it was isolated depended on the program of chemotherapy.

Journal of Investigative Dermatology, Baltimore

8:183-226 (April) 1947

Treatment of Mycosis Fungoides with Nitrogen Mustard. H. H. Henstell and J. N. Tober.—p. 183.
Method for Protection of Patients with Solar Urticaria. L. Rubin, P. L. Beal and S. Rothman.—p. 189.
Somatopsychic Medicine. C. H. Campbell.—p. 191.
Treatment of Penile Condylomata Acuminata with Podophyllin: Observations on Prevention of Balanitis. T. H. Finkle and E. J. Frishwasser.—p. 199.
Photosensitization in the Treatment of Psoriasis. E. Epstein.—p. 203.
Penicillin Ointments: Study of Effect of Ointment Ingredients on Stability of Penicillin. T. J. Macek, W. C. Gakenheimer and P. J. Daughenbaugh.—p. 209.
Exudative Discoid and Lichenoid Chronic Dermatosis (Sulzberger-Garbe): Histopathologic Study. W. Sachs and N. Kirsch.—p. 215.
Polyethylene Glycol Vehicles in Dermatology. M. T.-R. Maynard.—p. 223.

Journal of Lab. and Clinical Medicine, St. Louis

32:349-474 (April) 1947. **Partial Index**

Rapid Identification of Shigella in Public Health Laboratory. W. W. Ferguson, Mary Branston, Grace L. McCallum and Margaret J. Carlson.—p. 349.
*Serologic Response in Murine Typhus as Measured by Weil-Felix, Rickettsial Complement Fixation and Rickettsial Agglutination Reaction. C. T. Nelson.—p. 360.
Influenza B in Needham, Mass., December 1945: Serologic Studies in Cases and in Family Contacts. M. Finland, Mildred W. Barnes, M. Meades, E. M. Ory and A. D. Rubenstein.—p. 367.
Effect of Antihistamine Drugs on Serum-Induced Myocarditis in Rabbits. F. A. Kyser, J. C. McCarter and J. Stengle.—p. 379.
Evidence for Activity of Second Member of Vitamin M Group (Fermentation Factor) in Sprue: Case Report. E. Jones, H. F. Warden and W. J. Darby.—p. 387.
*Thiouracil and Propylthiouracil: Comparative Clinical Study. W. H. Beierwaltes and C. C. Sturgis.—p. 392.
Observations on Diurnal Excretion of Urobilinogen in Urine of Normal Subjects and Patients with Laënnec's Cirrhosis. E. Pellegrino, A. J. Patek Jr., Ann Colcher and Beatrice Domanski.—p. 397.
Comparison of Nutritive Value of Proteins in Mixed Diets for Dogs, Rats and Human Beings. D. M. Hegsted, Virginia Kent, A. G. Tsongas and F. J. Stare.—p. 403.
Prothrombin Time Studies on Human Beings and Experimental Animals. G. A. Nitshe Jr., H. W. Gerarde and H. F. Deutsch.—p. 410.
Two Popular Fallacies Regarding Rh: Preliminary Report of Some Thought Provoking Observations. P. H. Hattersley.—p. 423.

Serologic Response in Murine Typhus.—The studies reported by Nelson were undertaken primarily to investigate the course of antibody development in patients with murine typhus by comparing the results of the Weil-Felix test with the specific rickettsial agglutination and complement fixation reactions. It was hoped that in this way it might be possible to evaluate more clearly the relative usefulness of the murine rickettsial agglutination and complement fixation tests in the early diagnosis of the disease. Multiple samples of blood serum were collected over a period of several weeks from patients with clinical evidence of typhus infection. Almost all these patients were residents of communities in the Southeasten United States where murine typhus is endemic. The serologic response in 22 cases of murine typhus has shown that the Weil-Felix and rickettsial agglutination reactions usually become positive earlier than the rickettsial complement fixation test in this disease. As far as the detection of murine typhus in its acute stages in man is concerned, the relatively simple rickettsial slide agglutination test is fully as effective as the more elaborate complement fixation reaction.

Comparison of Thiouracil and Propylthiouracil.—A statistical comparison of 98 thyrotoxic patients treated with 0.6 Gm. of thiouracil per day and 42 treated with 0.15 Gm. of propylthiouracil per day revealed practically identical effective-

ness of the two drugs in these doses. The incidence of toxic reactions with propylthiouracil is much less than with thiouracil. Severe toxic reactions might be encountered if the present dosage of propylthiouracil is significantly increased. The available data indicate that 150 mg. is probably the most satisfactory maximum dosage of propylthiouracil.

Journal of Pediatrics, St. Louis

30:371-484 (April) 1947

Thiouracil in Childhood Hyperthyroidism. L. P. Williams and F. R. Janney.—p. 371.
*Infectious Hepatitis in Childhood: Report of Institutional Outbreaks and Comparison of Disease in Adults and Children. Dorothy M. Horstmann, W. P. Havens Jr. and Joyce Deutsch.—p. 381.
*Hypercalcemia and Idiopathic Hyperplasia of the Parathyroid Glands in Infant. E. L. Pratt, Betty B. Geren and E. B. D. Neuhauser.—p. 388.
Bone Marrow Infusion in Childhood: Experiences from 1,000 Infusions. S. Heinild, T. Søndergaard and F. Tudvad.—p. 400.
Estimation of Extracellular and Total Body Water in Newborn Human Infant with Radioactive Sodium and Deuterium Oxide. L. B. Flexner, W. S. Wilde, N. K. Proctor, D. B. Cowie, G. J. Vosburgh and L. M. Hellman.—p. 413.
Renal Glycosuria Associated with Pentosuria. I. Dunsky and Grace Lawrence.—p. 416.
Poisoning Due to Ingestion of Wax Crayons: Report of Case. J. A. Jones and H. Brieger.—p. 422.
Double Aortic Arch. Susanna Gordon.—p. 428.
Acute Meningococcemia. L. K. Sweet, H. F. Dowling and M. Jane Howell.—p. 438.
*Comparative Effect of Parenteral Administration of Single Massive Doses of Vitamin D in Oil-Ether Vehicle. H. Vollmer and B. L. Oser.—p. 446.
Capillary Fragility and Menses in Rheumatic Girls. E. E. Brown and Valentina P. Wasson.—p. 455.

Infectious Hepatitis in Childhood.—Horstmann and her associates describe two epidemics of infectious hepatitis in children. In one, 53 cases of hepatitis with jaundice occurred, along with 56 cases of questionable hepatitis without icterus. In the other institution, 68 patients developed hepatitis with jaundice; of these 44 were children. In all of the juvenile patients the disease was mild and of relatively short duration as compared with the disease in adults. The difference between the severity of infectious hepatitis in children and in adults is comparable to the difference which exists between the childhood and adult forms of the more common virus infections. Measles, mumps and chickenpox are all likely to be more severe and attended with more severe complications in the adult than in the child. The authors deplore that the mildness of infectious hepatitis in children has tended to cause the disease to be belittled. There is no evidence that such mild cases are less dangerous to the community from the infectious point of view than are the more severe adult cases.

Hypercalcemia and Hyperplasia of Parathyroid in Infant.—Pratt and his associates present clinical and laboratory investigations on an infant aged 10 months exhibiting extreme hypercalcemia, mild acidosis, impairment of renal functions and low urinary excretion of calcium. The postmortem examination revealed chief cell hyperplasia of all four parathyroid glands, nephrocalcinosis and osteitis fibrosa. The apparently unique combination of hypercalcemia and chief cell hyperplasia of the parathyroids was not satisfactorily explained.

Parenteral Administration of Vitamin D.—Vollmer and his co-workers describe experiments on rats which were carried out to determine the comparative therapeutic and prophylactic effect of a single massive dose of vitamin D_2 administered orally in oil, intramuscularly in oil and intramuscularly in oil-ether solution. The superiority of the oil-ether injection and the oil injection over the oral doses was statistically significant. The superiority of the parenteral vitamin D_2 administration in an oil-ether vehicle in the therapeutic as well as the prophylactic experiments seems to indicate quick absorption and satisfactory retention of the vitamin administered by this method. The inferiority of the parenteral administration of vitamin D_2 in oil in the therapeutic experiments may be ascribed to a delayed vitamin D absorption from the oily deposit in the tissues. The inferiority of the oral administration of vitamin D_2 in oil in the prophylactic experiments may be explained by incomplete absorption of massive doses from the gastrointestinal tract and loss of vitamin D with the feces. If observations in animals are applicable to infants and children, one may expect that intramuscular injection of a single massive dose of vitamin D_2 in an oil-ether vehicle will cure rickets at least as promptly as other methods of administration and is at least equivalent to the other methods in its prophylactic effect, giving complete protection against rickets for at least three months.

Michigan State Medical Society Journal, Lansing

46:377-488 (April) 1947

The Physician and the State. K. Sigler.—p. 425.
Operation of Public Law 725: Hospital Survey and Construction Act. H. E. Hilleboe.—p. 428.
Modern Influences in Medical Practice. T. P. Murdock.—p. 431.
Cardiac Complaints and Chronic Nonspecific Prostatitis. J. A. Winter.—p. 433.
*Treatment of Hyperthyroidism: Present Status. G. Crile Jr.—p. 437.
Paralysis of Larynx. L. H. Clerf.—p. 441.
Determination of Unmet Need for Medical Attention Among Michigan Farm Families. C. R. Hoffer and E. A. Schuler, in cooperation with Rosalie Neligh and T. Robinson.—p. 443.
Functional Bleeding at Different Age Periods: Significance and Management. E. Novak.—p. 447.
Functional Hyperinsulinism: Common and Well Defined Clinical Entity Amenable to Medical Management. J. W. Conn.—p. 451.

Treatment of Hyperthyroidism.—Crile thinks that the disappointment felt by the profession in thiouracil and thiobarbital has prejudiced many physicians against the antithyroid drugs and has led them to regard with suspicion the more recently developed thiouracil derivatives. This suspicion is poorly founded, because propylthiouracil has proved safe and effective. It accomplishes a physiologic rather than an anatomic thyroidectomy, and the mechanism by which it produces a remission is comparable in many respects to that of thyroidectomy. Propylthiouracil is the treatment of choice for diffuse goiter with hyperthyroidism. Thyroidectomy is seldom indicated. Because adenomatous goiters are tumors of the thyroid and because tumors are potentially dangerous, thyroidectomy is still the treatment of choice for most nodular goiters of significant size. The majority of patients with nodular goiters and mild hyperthyroidism are best prepared for operation with iodine. Propylthiouracil is used in the preoperative preparation of patients with severe hyperthyroidism, of patients over 50 and of patients with complications increasing the risk of operation. Nodular goiter with hyperthyroidism occurring in aging patients or in patients with short life expectancy can be successfully and definitively treated with propylthiouracil.

New England Journal of Medicine, Boston

236:523-554 (April 10) 1947

Use of Curare in Anesthesia. E. C. Allen and U. H. Eversole.—p. 523.
Experiences with Curare in Anesthesia. S. C. Siggin, P. E. Schultz and P. Saunders.—p. 526.
Carcinoma of Breast: Review of First Three Years' Experience in Cancer Section of Westfield State Sanatorium. F. S. Hopkins and N. Egnatz.—p. 530.
*Multiple Congenital Defects Following Maternal Varicella: Report of a Case. E. G. Laforet and C. L. Lynch Jr.—p. 534.
Effects of Protein Deficiency on the Pregnant Woman and Fetus and on the Infant and Child. H. C. Stuart.—p. 537.
Basal Cell Papilloma of Bronchus, with Cornification.—p. 542.
Carcinoma of Common Bile Duct.—p. 545.

236:555-610 (April 17) 1947

The Value of Imponderables. R. Fitz.—p. 555.
The Hospital—Look Ahead: Some Aspects of Clinical Investigation. G. R. Minot.—p. 563.
Atelectasis as a Factor in the Medical Treatment of Pleural Empyema: Necessity of Combating Atelectasis by Remedial Breathing Exercises. F. G. Fleischner.—p. 566.
Electrocardiography. L. B. Ellis.—p. 572.
Ewing Tumor of Fibula.—p. 599.
Acute and Chronic Salpingo-Oophoritis: Placental Polyp.—p. 601.

Congenital Defects Following Maternal Varicella.—Laforet and Lynch report that in an infant whose mother had varicella during the eighth week of pregnancy the following multiple congenital defects were present: talipes equinovarus on the right side, paralysis and muscular atrophy of the right lower extremity, defective development of the toes of the right foot, cortical atrophy, hypoplasia or aplasia of the cerebellum, bilateral optic atrophy, pilonidal sinus, torticollis to the right, cicatricial lesions of the skin of the left lower extremity, unde-

scended testis on the left and insufficiency of the anal and vesical sphincters. The episode of maternal varicella was well authenticated. Reviewing the present opinion on the pathogenesis of anomalies associated with maternal antepartum virus infection, the authors emphasize the possible teratogenic propensities of maternal virus diseases occurring early in pregnancy.

New Orleans Medical and Surgical Journal

99:485-544 (April) 1947

Socialized Medicine. F. P. Rizzo.—p. 485.
*Ligation of the Vena Cava: Critical Evaluation Based on a Study of 22 Cases. C. G. Collins, E. W. Nelson, J. R. Jones and others. —p. 488.
The Unstable Colon: Its Role as Precursor to Diverticulosis. E. A. Schexnayder.—p. 496.
Actinomycosis: Report of 12 Cases, with Special Reference to a Mediastinal Case. W. J. Hollis and M. D. Hargrove.—p. 499.
Multiple Operative Procedures in Case of von Recklinghausen's Disease. J. P. Woodhall and D. H. Echols.—p. 507.
Traumatic Rupture of the Pancreas: Case Report. G. McHardy, D. C. Browne and G. Welch.—p. 511.
Evaluation of Psychogenic Factor in Peptic Ulcer. S. J. Orloff.—p. 513.

Ligation of Vena Cava.—Collins and his associates say that ligation of the inferior vena cava and the ovarian veins has been done in 22 cases at Tulane University School of Medicine. They stress that ligation of the vena cava carries a low operative mortality. The end results and the appearance and function of the patients' extremities are good. The procedure is indicated in cases of phlebothrombosis of the iliac vessels whether the clotting process occurs preoperatively or postoperatively. It is also indicated in cases of postabortal and postpartal puerperal sepsis if conservative therapy is ineffective, if infarction to the lung develops or if the patient has an infarct to the lung from a suppurative pelvic thrombophlebitis when first seen. The minimal amount of edema and circulatory disturbance occasioned by vena cava ligation in the reviewed cases is due to the fact that at the time of operation the sympathetic chains are sectioned; if this is not possible at operation routine blocking of the lumbar sympathetics is performed, bilaterally, daily, for five or six days after operation.

New York State Journal of Medicine, New York

47:929-1056 (May 1) 1947

Pilonidal (Sacrococcygeal) Cyst and Sinus: Evaluation of Some Recently Proposed Therapeutic Measures. R. Turell.—p. 977.
Use of Medicament to Reduce Appetite in Treatment of Obesity and Other Conditions. W. L. Gould.—p. 981.
*Office Procedure in Applying Penicillin Therapy to Acute Suppurative Bartholinitis. M. A. Goldberger and L. S. Lapid.—p. 984.
Pathogenesis of Spina Bifida and Related Congenital Malformations. A. S. Wiener.—p. 985.
*Symptomatic Treatment of Hay Fever with Diaminobenzopyridyl Hydrochloride (N'-Pyridyl-N'-Benzyl-N-Dimethylethylenediamine Hydrochloride), (Pyribenzamine). H. Leibowitz, I. M. Kurtz and E. Schwartz. —p. 989.
Nitrogen Mustards and Application in Neoplastic Diseases. D. A. Karnofsky.—p. 992.
Pentothal Nitrous Oxide-Oxygen Anesthesia. E. H. Bachrach.—p. 994.
Coexistence of Malformations of Genital and Urinary Tracts in Women. W. T. Pommerenke and J. A. Benjamin.—p. 996.
Amphetamine Sulfate (Benzedrine) in Treatment of Obesity: Critical Estimate. H. B. Richardson.—p. 998.
Benzedrine Sulfate in Elderly People. W. Caveness.—p. 1003.

Office Procedure for Penicillin Therapy of Suppurative Bartholinitis.—Goldberger and Lapid aspirate the suppurating Bartholin abscess and inject penicillin directly into the cavity. Three patients so treated all complained of an exquisitely tender local vulvar mass, extreme difficulty in walking and sitting and generalized discomfort. They all experienced immediate relief. One of the 3 patients had to be reaspirated twenty-four hours after the initial procedure because of the reaccumulation of pus in the cavity; this patient received 100,000 units of penicillin. The other 2 patients received 200,000 units of penicillin in one injection. With this procedure patients are not subjected to a minor operative procedure of incision and drainage with its postoperative care and longer reparative process. Within forty-eight hours there is no evidence of an inflammatory process.

Pyribenzamine in Treatment of Hay Fever.—Leibowitz and his associates review observations on the use of pyribenzamine hydrochloride in patients with hay fever due to ragweed. Of 86 patients 21 were treated with pyribenzamine alone, 47 with desensitization and pyribenzamine, and 18 with ragweed desensitization alone. The majority of patients obtaining relief from pyribenzamine, either alone or in conjunction with ragweed desensitization, required only two tablets a day. Side reactions occurred in 25 of the 68 patients receiving pyribenzamine. These effects disappeared gradually while the drug was still being administered. Pyribenzamine did not affect the blood count. The highest percentage of relief was obtained when pyribenzamine was administered together with ragweed desensitization (35 of 47). Pyribenzamine alone produced relief in 9 out of 21 cases. Ragweed desensitization alone relieved 10 out of 18 cases.

North Carolina Medical Journal, Winston-Salem

8:191-266 (April) 1947

*Spherocytosis and Increased Erythrocyte Fragility in Four Generations of Family of Patient with Congenital Hemolytic Anemia. C. N. Herndon and J. B. Alexander.—p. 196.
Amebiasis: Case Reports from North Carolina Baptist Hospital. J. W. Abernethy and D. Cayer.—p. 201.
Peptic Ulcer of Duodenum: Review of 98 Cases Treated Surgically. H. H. Bradshaw and F. Hightower.—p. 204.
Treatment of Meningitis in Childhood, with Particular Reference to Use of Penicillin and Streptomycin. R. B. Lawson and W. M. Kelsey. —p. 211.
Regional Enteritis, with Observations Concerning Its Pathogenesis. R. P. Morehead.—p. 216.
*Use of BAL in Treatment of Heavy Metal Poisoning. C. R. Welfare. —p. 219.
Special Disabilities in Children with Organic Brain Pathology. L. J. Thompson.—p. 224.
Basis for Therapy in Shock. H. D. Green.—p. 228.
Renal Tumors. C. M. Norfleet Jr.—p. 236.

Hemolytic Anemia.—Herndon and Alexander studied the family of a woman aged 39 with congenital hemolytic anemia. Determinations of the hemoglobin red cell count, erythrocyte fragility tests and wet smear examinations for spherocytes were performed on 15 relatives in four generations. Six of these relatives presented definite laboratory evidence of defective erythrocytes, only 1 of them gave a history of jaundice, and the remaining 5 were reported to be in good health. Determinations were made of the blood groups, blood types and Rh factor in 16 members of the family and of the taste reaction to phenyl-thio-carbamide in 12 members of the family. Although this series is too small to be of significance in linkage calculations, the data are presented in the hope that additional similar data may become available. It is suggested that congenital hemolytic anemia is due to the action of a simple dominant gene, with variable expressivity. Emphasis is put on the fact that history alone is entirely unreliable in classifying relatives of a patient with congenital hemolytic anemia as normal or affected. The variability in the clinical picture is so great that accurate hematologic examination is necessary for proper classification of even apparently normal relatives.

BAL in Heavy Metal Poisoning.—According to Welfare there is evidence that arsenic produces intoxication in man and animals by depressing intracellular tissue respiration. It produces this depression by attaching itself to sulfhydryl radicals of enzymes responsible for the metabolism of fats and carbohydrates, thus interfering with the function of these enzyme systems. British Anti-Lewisite (BAL), a dithiol (2,3-dimercaptopropanol), by supplying more readily available sulfhydryl groups, is able to remove arsenic from the enzyme systems and thereby reactivate tissue respiration. BAL is a potent antidote for poisoning by other heavy metals such as mercury producing toxic effects in a manner similar to that of arsenic, and BAL is probably useful in the treatment of poisoning by bismuth, gold and antimony. On the basis of animal experiments it would appear that BAL is contraindicated in cases of poisoning by lead, thallium and selenium. While best results in heavy metal poisoning are obtained by early treatment with BAL, the compound was also effective when treatment was started three weeks after the appearance of erythema with a scaling eczematoid process and edema in a pregnant woman aged 39 with syphilis to whom mapharsen had been given; complete recovery resulted from the administration of a total dose of 3,750 mg. of BAL.

Philippine Medical Association Journal, Manila

22:449-510 (Nov.) 1946

Transurethral Resection in Treatment for Prostatic Median Bar. L. F. Torres Jr. and P. Morales.—p. 449.
Management of Prolapse of Uterus. C. Manahan and J. Coronado. —p. 453.
Epidermoid Carcinoma of Vulva: Radical Vulvectomy. M. C. Magboo. —p. 457.
*Is Hypoproteinemia the Cause of the Toxemias of Pregnancy? H. Acosta-Sison and A. Infantado.—p. 459.

Hypoproteinemia and Toxemias of Pregnancy.—Acosta-Sison and Infantado determined the serum protein values of 168 consecutive obstetric cases to ascertain the incidence of hypoproteinemia, also the correlation between hypoproteinemia and edema, hypertension and albuminuria. They stress that there is a high correlation between hypovitaminosis and hypoproteinemia. Though there is high correlation between edema and hypoproteinemia, still there are cases with edema which do not show hypoproteinemia; and many cases of hypoproteinemia are without edema. There is no correlation between hypertension and hypoproteinemia. Though nearly half the cases of preeclampsia and eclampsia show hypoproteinemia, it is probably not the cause of preeclampsia and eclampsia. Rather they are the result of the toxic condition present. For if hypoproteinemia were the cause, there would be a high incidence of these toxemias in hypovitaminosis, beriberi or hydatidiform mole, in all of which the incidence of hypoproteinemia is high.

Psychosomatic Medicine, Baltimore

9:77-150 (March-April) 1947

*Psychologic Invalidism in Thyroidectomized Patients. J. Ruesch, Carole Christiansen, L. C. Patterson and others.—p. 77.
Psychosomatic Medicine: Somatization Reactions. W. C. Menninger. —p. 92.
Brief Psychotherapy in Psychosomatic Problems. R. R. Grinker.—p. 98.
"Ill Health" as an Expression of Anxiety in a Combat Unit. R. Kaufman.—p. 104.
Central Angiospastic Retinopathy: Psychosomatic Study of Its Occurrence in Military Personnel. M. A. Zeligs.—p. 110.
*Phantom Limb. J. R. Ewalt, G. C. Randall and H. Morris.—p. 118.

Psychologic Invalidism in Thyroidectomized Patients.—Ruesch and his associates review the results of personality study on 43 patients who had been subjected to subtotal thyroidectomy and were selected from the material of the thyroid clinic of the University of California Hospital in such a manner as to constitute a fair representation of the several thousand patients followed in the clinic. The authors believe that patients who are afflicted with thyroid disease are not entirely normal as far as sociopsychologic aspects are concerned. The group divides into two distinct entities: the normal or almost normal girl and the hysterical or anxiety type. While surgical procedures are tolerated exceedingly well by the normal girls, delayed recovery is almost a probability in anxiety types and hysterical characters. In men the problem is similar, but instead of hysterical personalities one deals with dependent individuals. In preparation for surgery, psychiatric interviews might be helpful in spotting personality deviations of the anxiety, hysterical or dependent type. Therapeutic interviews before and after operation might improve the chances of recovery of patients with personality disorders.

Phantom Limb.—The report presented by Ewalt and his associates is based on observations on amputees during their stay in an army general hospital. Of a series of 2,284 patients with amputations, 404 had detailed psychiatric study. Phantom sensation has been found to occur in practically all individuals who are studied carefully. Spontaneous complaints of pain were rather rare among the group. The patients complaining of phantom pain were persons who had psychopathologic manifestations complicating the orthopedic picture. The authors gained the impression that phantom pain is merely the interpretation of a phantom sensation by certain psychopathologic persons. Two cases are reported which support this view. In both the pain tended to come and go with psychopathologic symptoms irrespective of what type of external treatment was carried on.

Public Health Reports, Washington, D. C.

62:565-595 (April 18) 1947

*Improved Method of Producing Smallpox Vaccine of Low Bacterial Content. D. H. Ducor.—p. 565.

Improved Method of Producing Smallpox Vaccine.—Ducor of the Laboratories of the Michigan Department of Health presents an improved method for the manufacture of smallpox vaccine of low bacterial content. Rigid attention to the details of sanitation during the quarantine and handling of animals, together with the treatment of operative surfaces with Roccal solution, constitute the improvements. With these improvements it is possible to eliminate the long storage period otherwise required for the destruction of bacterial contaminants in the vaccine. Roccal, which is a quaternary ammonium compound, is compared with brilliant green as a germicide. These germicides are applied to the skin surface of the operative field. Roccal was found to be a more efficient germicide with no demonstrable effect on the virus.

Radiology, Syracuse, N. Y.

48:319-440 (April) 1947

Reasons for the Common Anatomic Location of Pulmonary Tuberculosis. W. Dock.—p. 319.
Roentgen Findings in Early Coccidioidomycosis. H. W. Jamison and R. A. Carter.—p. 323.
Pulmonary Sarcoidosis: Early Roentgen Findings. L. H. Garland. —p. 333.
*Roentgen Therapy in Hodgkin's Disease. T. B. Merner and K. W. Stenstrom.—p. 355.
Early Effects of X-Rays on the Ovaries of Rat. L. Halberstaedter and M. Ickowicz.—p. 369.
Roentgenkymographic Study of Heart in Myasthenia Gravis. R. C. Batt.—p. 374.
Roentgen Findings in Torulosis: Report of 4 Cases. R. R. Greening and L. J. Menville.—p. 381.
Study of 24,615 Separation Chest Roentgenograms. B. J. O'Loughlin. —p. 389.
Aortic Aneurysm Secondary to Coarctation: Report of a Case Showing Calcification. S. B. Clark and E. C. Koenig.—p. 392.
Problems of Acquired Radioresistance of Cancer: Adaptation of Tumor Cells. F. Windholz.—p. 398.

Roentgen Therapy in Hodgkin's Disease.—Merner and Stenstrom review observations on 185 patients in whom Hodgkin's disease had been proved by biopsy. Multiple areas are involved by Hodgkin's disease throughout the body, but the peripheral lymph nodes are most frequently affected, followed by mediastinal nodes, abdominal nodes, spleen and bones. The disease is most common in the third decade, and about two thirds of the patients are males. The authors believe that the method of irradiation developed at the University of Minnesota Hospitals is highly efficient in the treatment of the lymphoblastomas. Treatment is considerably more intensive than in most series reported. A full course of therapy is applied to each area of involvement, the dosage varying between 1,000 and 2,000 tissue roentgens over a period of fourteen days. Large masses are treated more intensively, and the group of enlarged nodes causing the greatest distress is treated first. The five year survival rate was 21 per cent and the ten year rate 8 per cent. Other statistics have also been presented, demonstrating the increase in the survival period from the onset of symptoms in treated cases as compared with a series of untreated cases of lymphoblastoma reported by Minot in 1926. The unpredictable nature of Hodgkin's disease is recognized and claims of cure cannot be made. Roentgen therapy has greatly increased the survival rate.

Rhode Island Medical Journal, Providence

30:165-240 (March) 1947

Heart Disease and Employment. L. J. Goldwater.—p. 179.
Residency and Fellowship Program at R. I. Hospital: Introduction. A. M. Burgess.—p. 182.
Comparison of Volumes of Blood, Plasma and Circulating Cells in Secondary Polycythemia and in Severe Anemia. W. J. H. Fischer Jr.—p. 185.
Case of Corneal Ulcer Treated with Penicillin Solution. R. R. Chace. —p. 187.
Our Latest Endeavor—Microfilming. Elizabeth Bingham.—p. 212.
Hospitals in Postwar World. C. R. Metcalf, R. W. Tuttle and R. W. Robinson.—p. 224.

30:241-324 (April) 1947

Xeroderma Pigmentosum. F. Ronchese.—p. 255.
Medicine in the Time of the Crusades. R. Hammond.—p. 258.
Water Pollution Control. A. D. Weston.—p. 263.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

Australian and New Zealand J. Surgery, Sydney

16:163-232 (Jan.) 1947

*Penicillin in Treatment of Infections in Nasal Passages and Sinuses. R. E. Dunn.—p. 163.
Nature of Pilonidal Sinus. E. S. J. King.—p. 182.
Drainage in Infected Cavernous Sinus Thrombosis. W. A. Fairclough. —p. 193.
Preliminary Note on Study of Postoperative Thrombosis, with Special Reference to Prethrombotic State. H. Cummine.—p. 197.
Intrathoracic Neurogenic Neoplasms, with Report of 4 Cases. M. P. Susman.—p. 200.
Bronchoscopy in Postoperative Pulmonary Complications. H. B. Harwood.—p. 206.

Penicillin in Nasal Infections.—Dunn presents an account of an attempt to treat infective conditions of the nose and accessory sinuses with penicillin. The selection of cases depended on the presence of a penicillin sensitive organism in the antral washes or nasal discharge, while the methods of administration were determined by the most efficacious means of delivering penicillin to the regions involved. Administration was local, intramuscular or oral or a combination of these methods. The majority of patients treated with penicillin claimed relief from symptoms and showed improvement on x-ray examination. The greater proportion of these would have responded to ordinary conservative treatment. Three patients from whose antral washes a pure penicillin sensitive organism was grown before treatment grew a culture of Bacillus pyocyaneus after prolonged penicillin treatment. Three patients developed a rash in the vicinity of the nasal vestibule, which cleared after the cessation of penicillin treatment. Penicillin counteracted secondary infection of the nasal passages following a common cold. In sinusitis only x-ray investigation can decide which method of applying penicillin would be most efficacious. Penicillin, helpful in the treatment of acute and subacute exacerbations, cannot produce permanent relief in the presence of gross chronic infective mucosal changes; nor can patients with antro-oral fistula, foreign body in the antrum or conditions producing stasis in the nasal passages or accessory sinuses hope for freedom from recurrence of infection if penicillin treatment alone is undertaken.

British Journal of Urology, London

19:1-60 (March) 1947

Abacterial Pyuria from New Angle. G. H. Baines.—p. 6.
Retrocaval Ureter, with Description of an Operation Case Complicated by Ureteric Calculus. J. N. Young.—p. 22.
Pyelographic Test for Renal Fixity During Respiration. F. Martino Savino.—p. 29.
Reiter's Disease Followed by True Infective Abacterial Pyuria. A. E. Goldstein.—p. 32.

British Medical Journal, London

1:517-552 (April 19) 1947

Louis Pasteur. A. Fleming.—p. 517.
Effects of Certain Diets on Loss of Nitrogen in Urine After Experimental Burns. E. A. Sellers and C. H. Best.—p. 522.
Rations and Nutritional Needs. E. R. Bransby and H. E. Magee. —p. 525.
Simple Formula for Calculation of Atmospheric Dryness. R. L. Wynne. —p. 528.
Traumatic Uremia: Recovery. J. C. Scott and C. G. Rob.—p. 529.
*Massive Penicillin Doses in General Practice. D. P. Wheatley.—p. 530.

Massive Doses of Penicillin in General Practice.—Wheatley cites histories of 7 patients with conditions commonly encountered in general practice who responded well to large daily doses of penicillin. The first patient was a woman who had been troubled for some time with recurrent and severe boils in the nose. The usual duration of one of these was one to two weeks. When she consulted the author she had a full blown and painful boil just inside her nose. She was given 200,000 units of penicillin by intramuscular injection, this being repeated the following day. The next day the boil had almost completely subsided. The other patients had whitlow, carbuncles or purulent conjunctivitis. Carbuncles cleared up in about five days and the patients were fit for work within a week. In none of the patients was local treatment given after penicillin therapy was started. The 1 patient who was given penicillin twice daily fared no better than the others who received only one daily dose.

1:553-584 (April 26) 1947

*Vitamin A in Infective Hepatitis. A. D. Harris and T. Moore.—p. 553.
Tuberculous Pericarditis. H. Kopelman.—p. 559.
Medullary Suprarenal Chromaffinoma Producing Malignant Hypertension. D. Gutmann.—p. 563.
Case of Pheochromocytoma. J. M. K. Spalding.—p. 564.
Seminal Fluid Acid Phosphatase in Sterility. G. E. Delory.—p. 566.

Vitamin A in Infective Hepatitis.—In preliminary experiments defective dark adaptation was used as an indication of vitamin A abnormality. Chemical methods were used in investigation made on 32 military patients, of whom 30 had infective hepatitis and 2 jaundice following arsenotherapy. Harris and Moore stress that in the early stages of infective hepatitis the vitamin A content of the plasmas was often low. In estimations by the antimony trichloride method, values of 30 international units per hundred cubic centimeters or less were sometimes observed, as compared with an average of 118 international units for normal subjects. The low plasma vitamin A seems an adequate cause of the impairment of dark adaptation in this disease. The plasma vitamin A increased during convalescence. Variations in the carotenoid content of the plasma were similar to those found for vitamin A, but the return to normal was less rapid. The changes in plasma vitamin A and carotenoids went roughly parallel with the prothrombin value and with hippuric acid tolerance and moved in an opposite direction to the plasma bilirubin. In "absorption tests" with massive doses of vitamin A it was found that in the early stages of infective hepatitis substantial amounts of vitamin A were excreted in the feces, and sometimes there were traces in the urine. Excretion was not found in normal subjects given the same doses or in the same patients during convalescence. In some cases the plasma vitamin A failed to show the usual rapid increase after massive dosing. It appears that in infective hepatitis the mechanisms responsible for the absorption of vitamin A from the intestine, for its deposition in the liver, and for its release into the blood stream are not always involved to the same degree. The interpretation of the results of "absorption tests" as a measure of the degree of injury to the liver therefore seems uncertain.

Edinburgh Medical Journal, London

54:65-128 (Feb.) 1947

Etiology and Diagnosis of Amebiasis. R. M. Murray-Lyon.—p. 65.
Amebiasis. J. D. S. Cameron.—p. 76.
Outlet Contraction of the Pelvis.—W. I. C. Morris.—p. 90.
Care of the Small Premature Infant. H. L. Wallace.—p. 111.
*Common Physical Signs in Old Age. T. H. Howell.—p. 123.

Common Physical Signs in Old Age.—Howell discusses some of the physical signs encountered during a survey of 340 healthy pensioners. He stresses that many physical signs are quite compatible with an active and healthy old age. A high blood pressure, cardiac murmurs, thickened arteries, absent tendon reflexes, some pigmentation of the skin and a lowered temperature are all to be found in normal old people.

Glasgow Medical Journal

28:103-126 (April) 1947

*Dermatitis as a Monosymptomatic Manifestation of Nicotinic Acid Deficiency. A. J. S. McFadzean.—p. 103.
Tomography of Bronchiectasis by Means of Preselected Planes of Section. E. Tisceneo.—p. 116.

Dermatitis and Nicotinic Acid Deficiency.—McFadzean directs attention to skin lesions attributable to nicotinic acid deficiency unassociated with other symptoms of such deficiency and occurring in otherwise normal persons. The lesions were seen in early summer in the Middle East in 62 men of European stock. The 44 cases making up the first group occurred in a closed community of 2,764 men on the same diet and living under identical conditions. Group 2 consists of cases seen in the wards or outpatient department of a Middle East general hospital. All stages in development of the characteristic skin

lesions of pellagra were seen, varying from the acute erythema with edema, vesiculation and crusting to the classic hyperpigmented, hyperkeratotic plaques with desquamation. Clinically the skin lesions were consistent with the dermatitis of pellagra, and consideration of the distribution of the lesions and the relevant circumstances led to the hypothesis that they had been provoked by solar irritation. These lesions could be reproduced experimentally by exposure to sun or to ultraviolet radiation and could be prevented or cured by large doses of nicotinic acid. Observations suggested that the local manifestations of nicotinic acid deficiency depended on local irritation; in the absence of this, no lesion developed. A method of provoking dermatitis by ultraviolet radiation has been described and should prove of diagnostic value in subclinical or suspected nicotinic acid deficiency.

J. of Neurol., Neurosurgery and Psychiatry, London

9:113-162 (Oct.) 1946

Collagen Content of Rabbit Sciatic Nerve During Wallerian Degeneration. M. Abercrombie and M. L. Johnson.—p. 113.
Neurotic Predisposition and Disorganization Observed in Experiments with Cambridge Cockpit. D. R. Davis.—p. 119.
Clinical and Pathologic Aspects of Progressive Atrophies of Globus Pallidus Alone or with Luy's Body. L. van Bogaert.—p. 125.
*A Case of Systemic Torula Infection with Tumor Formation in Meninges. L. Krainer, J. M. Small, A. B. Hewlitt and T. Deness.—p. 158.

Systemic Torula Infection with Tumor in Meninges.—According to Krainer and his associates, typical cases of torula meningoencephalitis show diffuse, patchy meningitis with perivascular and embolic extension into the brain substance, while tumor formation in the meninges is rare and systemic lesions are usually inconspicuous. The causative organism is a spheroidal, encapsulated, yeastlike fungus. The case described in this paper concerned a Sikh, aged 32. It showed features distinct from the common clinical and pathologic picture of torula infection. Systemic lesions were prominent before signs of central nervous system involvement. The lesion in the cranium gave rise to the clinical picture of a space occupying lesion situated in the left frontal region. At operation a globular tumor was found sharply demarcated from the brain substance and attached to the dura mater, the consistency and color being that of a meningioma. The microscopic examination showed the typical picture of torula granuloma. Specific cerebrospinal fluid changes were absent before and after operation. The causative organism was isolated from the systemic lesion of the right thigh before there were signs of a cerebral tumor. The organism was classified as type III of Torula histolytica Freeman.

Journal of Physiology, Cambridge

106:1-112 (March 15) 1947. **Partial Index**

Potentiating Action of Acetylcholine on That of Adrenalin. R. J. S. McDowall.—p. 1.
Effects of Magnesium Ions and of Creatine Phosphate on Synthesis of Acetylcholine. W. Feldberg and Catherine Hebb.—p. 8.
Effects of Analeptics on Fatigued Subject. D. P. Cuthbertson and J. A. C. Knox.—p. 42.
Binocular Summation Within Nervous Pathways of Pupillary Light Reflex. L. C. Thomson.—p. 59.
Subthreshold Potentials in Medulated Nerve. B. Katz.—p. 66.
Nervous Pathways of Intestinal Reflexes Associated with Nausea and Vomiting. R. A. Gregory.—p. 95.
Action of Soya Bean Trypsin Inhibitor as Antithromboplastin in Blood Coagulation. R. G. MacFarlane.—p. 104.

Journal of Tropical Medicine and Hygiene, London

50:61-78 (April) 1947

Kwashiorkor (Malignant Malnutrition) Arising in Assam. K. P. Hare.—p. 63.
Tuberculous Pericarditis in the African. M. Gelfand.—p. 68.
Tropical Pyomyositis. R. T. Burkitt.—p. 71.
*Chang Shan in Treatment of Malaria. Cheng Fang Tsu.—p. 75.

Chang Shan in Malaria.—According to Cheng Fang Tsu, the indigenous Chinese drug chang shan has been used in China for the treatment of malaria for more than two thousand years. The vomiting produced by the extract prepared according to the old methods has been largely eliminated by a newer method of extraction. The extract was given to 322 patients with benign tertian malaria; 67 per cent of these were cured, and of 253 patients with malignant tertian malaria 63 per cent were cured. A further series of 67 patients with tertian and 14 with malignant malaria were hospitalized and treated with chang shan. In most cases the chang shan extract was given by mouth, but in a few cases intramuscular injection was tried. The local reaction was not unduly severe. Gastric symptoms could be avoided by preliminary administration of bismuth subnitrate or hyoscyamus and sodium bicarbonate. So far no other secondary effects have been observed even when the individual dosage was raised to 8 cc. and the daily dosage to 40 cc.

Lancet, London

1:507-542 (April 19) 1947

Recovery from Starvation. R. O. Murray.—p. 507.
*Folic Acid in Treatment of Sprue Syndrome. L. S. P. Davidson, R. H. Girdwood and Elizabeth M. Innes.—p. 511.
Care of Urinary Tract in Paraplegic Patients: Review of 82 Cases. H. Donovan.—p. 515.
Bilateral Congenital Dislocation of Radial Head. R. K. Magee.—p. 519.
*Toxic Effects of Thiourea, Thiouracil, Methyl Thiouracil and Aminothiazole in Thyrotoxicosis. Margaret E. Morgans.—p. 519.

Folic Acid in Sprue.—Davidson and his associates report the hematologic changes in 10 cases of the sprue syndrome after treatment with synthetic folic acid. The hematologic response was considered unsatisfactory in 9 patients in that the treatment either produced no significant rise in the red cell count or failed to restore the blood level and hematologic picture to normal. Diarrhea was controlled and clinical improvement resulted in the 7 adults with tropical sprue or idiopathic steatorrhea. In contrast, no significant benefit was observed in the 3 patients with celiac disease. Folic acid did not improve fat absorption, as estimated by fat balance tests, in 5 out of 6 cases of the sprue syndrome. The unsatisfactory hematologic responses were in striking contrast to the results obtained by Spies et al. and Garcia Lopez et al. in cases of tropical sprue. These workers laid down criteria for the selection of patients, one of which was that the marrow should be megaloblastic. Only 1 of the patients in the authors' group had megaloblastic marrow at the start of folic acid therapy, and it was this 1 patient who obtained the most dramatic initial hematologic response.

Toxic Effects of Thiourea, Thiouracil, Methyl Thiouracil and Aminothiazole in Thyrotoxicosis.—Morgans found that thiourea and aminothiazole have many toxic effects and both depress the granular cells of the blood. Their use in clinical practice is no longer justifiable. Thiouracil and methyl thiouracil also depress the granular cells of the blood, but during their use the incidence of other complications is much lower than with the other two drugs. Thiouracil and methyl thiouracil can be used with a reasonable degree of safety, provided the patients are carefully watched.

1:543-582 (April 26) 1947

Surgical Handicraft. H. Ogilvie.—p. 543.
*Survival of Variola Virus in Dried Exudate and Crusts from Smallpox Patients. A. W. Downie and K. R. Dumbell.—p. 550.
Sonne Dysentery and Nonspecific Gastroenteritis in a Hospital. L. Martin and M. M. Wilson.—p. 553.
Diagnosis of Epilepsy in Service Cases. T. B. S. Dick.—p. 555.
Therapeutic Uses of Khellin: Method of Standardization. G. V. Anrep, M. R. Kenawa, G. S. Barsoum and G. Misrahy.—p. 557.

Survival of Variola Virus in Dried Exudate and Crusts from Smallpox Patients.—Downie and Dumbell used chorioallantoic membranes of developing hen's eggs to detect the presence of virus in vesicle fluid and crusts from smallpox patients. By this means virus was found to survive in diluted vesicle fluid dried on glass slides for thirty-five days when exposed to daylight and for eighty-four days when kept in the dark at room temperature. Virus was recovered from different samples of crusts kept at room temperature for several months and from one specimen kept thus for over a year. The authors have isolated virus from the sweepings from the ward floor near a bed in which a child was convalescing from smallpox. They also cite outbreaks among laundry workers who handled bedclothes of smallpox patients.

Medical Journal of Australia, Sydney

1:289-321 (March 8) 1947

*Penicillin in Treatment of Discharge from Middle Ear. R. E. Dunn. —p. 289.
Treatment of Placenta Previa. J. Chesterman.—p. 293.
Peripheral Venous Thrombosis: Preventive Measures and Treatment. A. B. Jones.—p. 297.
Postoperative Thrombosis and Blood Coagulation Times. H. Cummine. —p. 302.

Penicillin for Discharge from Middle Ear.—The records of the cases of discharging ears reviewed by Dunn were collected at the 115th (Heidelberg) Military Hospital. Penicillin was applied locally by intramuscular injection and by intramuscular and intrathecal injection. The response of a discharging middle ear to the local application of penicillin occurred within three to four days, while with intramuscular injections of penicillin about five or six days were required for the production of a dry ear. Forty of 63 patients with a middle ear discharge were found to be harboring a penicillin sensitive organism. Thirty of the 40 patients with a discharge from the middle ear which yielded penicillin sensitive organisms on culture responded to penicillin treatment. The intramuscular injection of penicillin was found beneficial in (1) acute otitis media: (*a*) patients not responding satisfactorily to sulfamerazine by mouth, harboring a staphylococcus in their aural discharge and obviously suffering from mastoiditis; (*b*) patients unable to be treated with sulfamerazine; (2) aural discharge following a fracture of the skull. In intracranial complications of mastoiditis penicillin therapy was disappointing.

1:321-352 (March 15) 1947

Anticoagulant Liberated from Perfused Mammalian Liver. E. R. Trethewie, Joan B. Cleland and T. J. Pengelley.—p. 321.
*Treatment of Rheumatoid and Infective Arthritis by Sulfonamides, with Special Reference to "Proseptasine," Sulfadiazine and Sulfaguanidine. L. J. A. Parr and Eva A. Shipton.—p. 323.
Physiologic Basis of Modern Anesthesia. R. H. Orton.—p. 332.

Sulfonamides in Rheumatoid and Infective Arthritis.—Parr and Shipton experimented over a period of approximately nine years with small daily doses of the sulfonamides given for a long period with a view to ascertaining their effects in the so-called rheumatic diseases. A small dosage of three tablets per day (1.5 Gm.) was employed. In acute cases associated with fever, larger doses up to three or four Gm. per day may be employed. Of the complications the rash is highly irritable and possibly allergic in nature, but the changes in the hemopoietic system are the most important. The importance and significance of anemia, leukopenia and eosinophilia are stressed. Administration of liver and yeast are indicated to prevent their occurrence. The probable value of pyridoxine and folic acid is mentioned. The beneficial effect of the drugs is shown by the improvement in color, the disappearance of cyanosis, the relief or disappearance of pain, swelling and stiffness of the joints, an increase in the number of red blood cells and the improvement in the blood sedimentation rate. For patients who only partly respond to the sulfonamides, the authors recommend supplementary therapy such as liver extract, yeast, calcium gluconate, iron and perhaps gold therapy. The mode of action of the sulfonamides is discussed with regard to their interference with oxygen consumption, and the view is expressed that with the smaller doses there occurs a vital suppression of bacterial oxygen consumption and therefore of bacterial growth, enabling the body cells with their smaller metabolic demand to regain the mastery over infection. The importance of repeated blood counts is stressed.

1:353-384 (March 22) 1947. **Partial Index**

*Beneficial Effects of Yeast in Diabetes Mellitus. L. J. A. Parr and Eva A. Shipton.—p. 365.
Anesthesia for Upper Abdominal Surgery. I. A. Schalit.—p. 367.
Precautionary Measure Advisable in Certain Endurance Tests. W. J. Lawrence—p. 370.

Beneficial Effects of Yeast in Diabetes Mellitus.—Parr and Shipton give the histories of 4 patients who were treated with yeast. There was evidence of considerable recovery in some of the beta cells of the islands of Langerhans. One patient responded to treatment without insulin. Of the 3 patients receiving insulin, 1 experienced a 40 per cent recovery and another has been off insulin for about eighteen months. The third is showing a pronounced decrease in the blood sugar level and diminution of insulin requirements. Either brewers' or bakers' yeast can be used. The authors mention the ability of yeasts to control ketosis and make the suggestion that the increase of diabetes in civilized communities may be due to the lack of the vitamin B complex in food. The need of a better type of bread is stressed.

New Zealand Medical Journal, Wellington

46:75-166 (April) 1947

Some Observations on High Blood Pressure. F. H. Smirk.—p. 86.
Surgery of Attic Suppuration. A. Wardale.—p. 102.
*Visceral Zoster: Report of a Case. W. M. Porteous.—p. 106.
Hydatid Cyst of Lung of Unusual Size: Report of Fatal Case. I. A. M. Prior.—p. 109.
Respiratory Motor Phenomena of Conversion Hysteria. I. M. Allen, —p. 113.
Investigation of Epidemic Eye Disease in Fiji. W. J. Hope-Robertson and L. S. Talbot.—p. 122.
Some Impressions of Surgery in Scandinavia. D. G. Simpson.—p. 129.
Fainting and Shock. J. W. Tomb.—p. 131.

Visceral Zoster.—According to Porteous painful visceral disease, not directly involving dorsal nerve roots, is sometimes associated with zoster in corresponding or neighboring dermatomes. The skin lesions may precede, accompany or follow the onset of visceral symptoms. A man aged 66, who for many years had been subject to nausea and heartburn after dietary indiscretions, for some months had noticed occasional soreness deeply situated beneath the left rib margin lateral to the rectus muscle. Two weeks before admission he experienced severe, shooting, knifelike pains in the right shoulder and back of the right arm, and four days later a zoster eruption appeared in the distribution of the tenth left dorsal nerve. The significance of the occasional pains beneath the left ribs, which were noted for some months prior to admission, is uncertain. Possibly they indicate that gastric ulceration was already present. Vomiting and gastrointestinal hemorrhage began five days after the appearance of zoster, and thirteen days later there was another severe hemorrhage, with probable perforation of the stomach and localized peritonitis. The pain felt in the right arm at the onset indicates that cervical as well as thoracic segments were affected by the herpetic process, and this is evidence in favor of the view that this was a case of "primary" or "idiopathic" herpes zoster with resulting visceral lesions. Complete recovery occurred.

Quarterly Journal of Medicine, Oxford

16:1-46 (Jan.) 1947

*Hunger Edema. F. A. Denz.—p. 1.
*Edematous Syndrome of Nephritis, with Special Reference to Prognosis. J. B. Rennie.—p. 21.
Osteomalacia with Looser's Nodes (Milkman's Syndrome) Due to Raised Resistance to Vitamin D Acquired About the Age of 15 Years. R. A. McCance.—p. 33.

Hunger Edema.—Denz reports investigations on the plasma protein levels in a small number of cases of edema occurring among German civilian internees. Total plasma proteins are decreased in patients with edema. Mean values of 5.24 Gm. per hundred cubic centimeters of plasma in slight cases, 5.07 in moderate cases and 4.90 in severe cases were obtained as compared with 7.04 Gm. for a normal control group. Plasma-albumin values in 14 edema cases ranged from 2.58 to 3.81 Gm. The lowered plasma proteins are believed to result in the accumulation of edema that commences with the first lowering of the protein level. The view that there is a critical level of plasma protein for the production of edema is not accepted. Four patients with apparently cured edema were exercised with reappearance of the edema. Two patients with edema were exercised while on full diet with persistence of the edema until the patients were allowed complete rest. Daily plasma protein determinations on edema cases showed a diurnal variation which was probably due to variation in the degree of hydration of the blood. The inaccuracy of the clinical assessment of edema is illustrated by a patient showing minimal edema, who lost

8.5 liters of edema fluid, whereas another showing severe edema lost only 2 liters of edema fluid. Much excess fluid may be hidden in the deeper connective tissues of the mediastinum and abdomen.

Edematous Syndrome of Nephritis.—Rennie discusses the fate of patients with that form of Bright's disease characterized by persistent edema and heavy proteinuria. The authors review the course of the edematous syndrome in 29 patients and determine the functional state of the kidneys in the survivors. The observations extended over a period of sixteen years. None of the patients had a history of acute nephritis. In 1946, 13 patients were alive. Three appeared to be well in every respect seven, thirteen and fifteen years from the appearance of edema, and 2 six and fourteen years afterward showed a faint trace of albumin in the urine as the sole abnormality. Three patients, although free of edema, showed greater albuminuria and microscopic hematuria. Of the remaining 5, 3 were still edematous and 2, although free of edema, were showing increasing hypertension. Sixteen patients had died, 12 of uremia, 3 of intercurrent infection, and 1 after an intravenous injection of a mercurial diuretic. All three recoveries were noted in patients in whom hypertension, azotemia and hematuria were not observed. Hypertension was an early indication of a progressive lesion. Of 14 patients with hypertension when first seen 8 died of uremia, and only 1 of the remainder improved.

Annales d'Oto-Laryngologie, Paris

13:393-520 (Sept.-Oct.) 1946

Pulmonary Abscess and Penicillin. A. Lemariey and P. Hypeau.—p. 393.
Bronchoscopotherapy in Bronchopulmonary Tuberculosis and Particularly in Bronchial Stenosis. A. Soulas.—p. 405.
*Acute Nondiphtherial Stenosing Laryngitis in Children and Its Endoscopic Treatment. L. Hamon and H. Le Mercier.—p. 413.
Complicated Histories of Foreign Bodies in Bronchi. A. Soulas.—p. 436.

Acute Stenosing Laryngitis.—Hamon and Le Mercier report 25 cases of acute nondiphtherial, stenosing laryngitis in children. Diagnosis was made with the aid of direct laryngoscopy and established by laboratory examination. Staphylococci were demonstrated in the great majority of cases, streptococci were observed occasionally and pneumococci and enterococci in only 1 case. Local treatment consisted in aspiration and direct intubation under direct laryngoscopy. This simple method provides control by the eye, prevents trauma and secures appropriate caliber of the tube. Removal of the tube was made under laryngoscopic control. General treatment for combating or preventing bronchopneumonia and edema consisted in administration of 1 to 2 cc. of propidon (Delbet's vaccine), oral administration of 2 to 4 Gm. of sulfadiazine or parenteral administration of 2 to 4 Gm. of solufontamide (a sulfonamide preparation) for twenty-four hours. The patient may be placed in an oxygen tent. Antispasmodics proved effective during the hours following removal of the tube. All danger of asphyxia disappeared within forty-eight to seventy-two hours and complete recovery occurred in 24 cases, while death occurred in 1 case with false membrane complicated by bronchopneumonia and measles. The value of penicillin in acute stenosing laryngitis is controversial, but its effect was decisive in 2 cases complicated by bronchopneumonia.

Lyon Chirurgical, Paris

42:5-128 (Jan.-Feb.) 1947. **Partial Index**

Tumors of Bones and Traumas. L. Tavernier and C. Godinot.—p. 5.
*Injury of Ampulla of Vater in Course of Gastroduodenectomy for Ulcer: Curative Treatment. P. Santy and P. E. Duroux.—p. 13.
*Procaine Infiltration of Carotid Sinus: Experimental and Clinical Basis: Indications and Results. J. Creyssel, P. Suire and J. Sournia.—p. 23.
Retropubic Prostatectomy Operation of Terence Millin. J. Cibert, H. Cavailher and J. Perrin.—p. 40.
Sulfate of Beta-Phenylisopropylamine in Biliary Hypertension. P. Mallet-Guy and R. Jeanjean.—p. 47.
Grave Postoperative Accidents in Alcoholic Addicts. P. Goinard, J. Ferrand and P. Tardieu.—p. 56.
Ligation of Abdominal Aorta (in Connection with Traumatic Aneurysmal Hematoma of Left Internal Iliac Fossa). P. Rochet, P. Santy, G. Allègre and L. Jacquemet.—p. 63.

Injury of Ampulla of Vater in Gastroduodenectomy.—The possibility of injuring the choledochus and Wirsung's duct in the course of gastroduodenectomy is one of the arguments often and justly invoked in opposing exeresis of chronic ulcers that have deeply penetrated into the pancreas. Santy and Duroux show that this is a comparatively rare occurrence and that this risk can be accepted, because means are available to remedy it.

Procaine Infiltration of Carotid Sinus.—Creyssel and his associates point out that since 1940, when they reported their first experiences with procaine infiltration of the carotid sinus for traumatic shock, this therapeutic method has been repeatedly discussed in the literature. Infiltration of the carotid sinus has been employed in five groups of cases: traumatic shock, surgical shock, obstetric shock, collapse due to acute peritoneal syndromes and adrenal insufficiency. Experimental studies have not only demonstrated the prophylactic value of carotid infiltration against operative shock but have also thrown light on the physiologic action of the sinus. The authors list indications for its employment and suggestions with regard to conditions in which it might be employed, such as grave craniocerebral trauma, epileptic attacks, cerebral apoplexy, laryngeal spasms and syndromes of parathyroid insufficiency.

Presse Médicale, Paris

55:173-184 (March 15) 1947

Segmental Bronchitis. E. Rist, P. Ameuille and J. M. Lemoine.—p. 173.
*Repeated Syncopes Connected with Sinus Hyperreactivity: Cured by Carotid Sinus Denervation. L. Leger and M. Coste.—p. 174.
Physiologic Effects of Oxidized Derivatives of Epinephrine. Z. M. Baco.—p. 175.

Carotid Sinus Denervation for Syncopes Caused by Sinusal Hyperreactivity.—Leger and Coste present the history of a man aged 62 who had repeated syncopal attacks, which usually came on in connection with some effort, such as rising from the bed. The complete ineffectiveness of various treatments together with the uncertainty of the diagnosis made one of the authors think of the possibility of a sinusal hyperreactivity, and tests which employed compression of the carotid sinus confirmed this hypothesis. Injection of procaine into the area of the carotid sinus in the course of a syncopal attack arrested the attack, and when the sinus was compressed under procaine no syncopal attack resulted. A carotid sinus denervation was performed, and since then there has been no recurrence of the attacks. In about two thirds cure resulted and in half of the remaining patients there was some improvement.

55:185-200 (March 19) 1947

*Experimental Research on Production of Ulcer and Gastric Perforation by Histamine. Pasteur Vallery-Radot, B. N. Halpern and J. Martin.—p. 185.
Present Role of Penicillin in Treatment of Syphilis. L. M. Pautrier.—p. 187.
Cutaneous Reaction to Local Sulfonamide Therapy (Particularities of Cutaneous Tests). A. Tzanck, E. Sidi and J. Roujeau.—p. 188.
Polypoid Formation of Gastric Mucosa with Umbilicated Summit Simulating Polyadenoma. G. Albot, L. Leger and A. Tricard.—p. 190.
Intraepiploid Inflammatory Pseudotumor Caused by Ascaris. J. Thibouméry.—p. 192.
Technic of Total Intraisthmian Hysterectomy. C. Elbaz.—p. 194.
Electroencephalogram in Cerebral Tumors. O. Sager and A. Kreindler.—p. 195.
Congenital Bronchiectasis. P. Lagèze and P. Buffard.—p. 197.

Histamine Produced Ulcer and Gastric Perforation.—Pasteur Vallery-Radot and his co-workers demonstrated that guinea pigs will tolerate 1,500 fatal doses of histamine, provided dimethyl-amino-ethyl-thiodiphenylamine (3015 RP) or dimethyl-amine-2-propyl-1-thiodiphenylamine (3277 RP) is given prophylactically. These synthetic antihistamines do not inhibit the stimulating secretory effect of histamine on the gastric mucosa, although they neutralize the effect of histamine on all the other physiologic functions. The authors produced various lesions in the gastric mucosa by intravenous injections of 100 to 1,000 fatal doses of histamine bichlorine hydrate. Microscopic examination revealed simple erosions, acute and indolent ulcers and perforation of the gastric mucosa, depending on the dose of histamine injected, on the interval between the injection and the time the animal was killed, varying from one hour to four days, and on spontaneous death resulting from repeated histamine injections. The experimental lesions were identical with those observed in man. Vascular involvement was limited to simple vasodilatation.

Rivista di Clinica Pediatrica, Florence

44:721-792 (Dec.) 1946. **Partial Index**

*Jaksch-Hayem-Luzet's Anemia. G. Careddu.—p. 721.

Jaksch-Hayem-Luzet's Anemia.—Careddu reports the occurrence in 2 infants aged 20 and 16 months, respectively, of Jaksch-Hayem-Luzet's splenic anemia which became manifest in early infancy and appeared to be caused by chronic suppuration of the scalp. Syphilis, tuberculosis, leishmaniasis and malaria were ruled out. Brothers and parents were normal. The differential diagnosis between this type of splenic anemia and Cooley's and Di Guglielmo's form is facilitated by the difference in fragility and the size of the erythrocytes. Jaksch-Hayem-Luzet's anemia is rare. The 2 reported cases were seen in a group of 50 cases of anemia in children.

Arch. Urug. de Med. Cir. y Especialid., Montevideo

29:221-332 (Sept.) 1946. **Partial Index**

*Diphtheria in Uruguay. C. Fabini and A. Panizza Blanco.—p. 221.
Technic for Determination of Blood Group of Hemolyzed Blood. H. Mazzella.—p. 268.

Diphtheria in Uruguay.—A review of the epidemiology of diphtheria in Uruguay before and after the establishment of obligatory vaccination of school children shows that morbidity and mortality indexes from diphtheria have greatly diminished in children in the age group 6 to 14 years, have moderately diminished in infants and children in the preschool age as a result of vaccination (although it is not as yet obligatory for children in this age group) and have not changed in infants in the first six months of life and in adults (age groups of nonvaccinated persons). At present acute diphtheria occurs in children who have had no vaccination and in those who had either incomplete immunization or had the vaccination three years or more before the actual epidemic. Obligatory vaccination of infants and children from the ages of 6 months up to 12 years and periodic revaccination are of importance in the prevention of diphtheria.

Medicina, Mexico, D. F.

27:149-172 (April 10) 1947. **Partial Index**

*Foot and Mouth Disease in Man. M. M. Arocha.—p. 163.

Foot and Mouth Disease in Man.—In the 5 cases reported, the clinical diagnosis was confirmed by laboratory tests (inoculations in guinea pigs). Four of the cases (2 acute and 2 subacute) were in 2 brothers and their parents; the fifth was in a friend of the children. Contagion seemed to be due to the proximity of the children to infected cows in their neighborhood.

American Review of Soviet Medicine, New York

4:291-384 (April) 1947

Medical Research in the U. S. S. R. V. Parin.—p. 292.
Research Program of the U. S. Public Health Service. R. E. Dyer. —p. 300.
Health in Byelorussia—A Devastated Area of the Soviet Union. L. A. Falk.—p. 306.
Microbiology in the Soviet Union. S. A. Waksman.—p. 314.
Experimental Cancer Research in the U. S. S. R. R. E. Kavetski. —p. 322.
Radiotherapy of Malignant Tumors in the U. S. S. R. M. Pobedinski. —p. 328.
Cancer as a Systemic Disease. C. P. Rhoads.—p. 333.
Cancer Prevention and Control in the U. S. S. R. A. I. Serebrov. —p. 343.
Organization of a Cancer Research Institute. S. P. Reimann.—p. 348.

Maandschrift voor Kindergeneeskunde, Leyden

15:1-35 (Jan.) 1947

*Toxoplasmosis: A Parasitologically Demonstrated Diagnosis (?); Significance of Clinical Manifestation in Infantile Toxoplasmosis. C. D. Binkhorst.—p. 1.
Desquamative Erythrodermia (Leiner-Moussous' Disease). C. van Waalwijk van Doorn.—p. 12.
Active Immunization Against Typhoid, Spotted Typhus, Bacillary Dysentery, Tetanus and Influenza. G. M. H. Veeneklaas.—p. 20.

Toxoplasmosis.—Binkhorst reports the probable occurrence of toxoplasmosis in a girl aged 6 years. Many signs characteristic of infantile toxoplasmosis were present, such as psychomotor disturbances, roentgenographically demonstrable intracranial calcifications, microphthalmia and chorioretinitis. The clinical diagnosis was made more probable by the demonstration of toxoplasma-like bodies in the cerebrospinal fluid. This is the first case reported in European literature in which diagnosis was made *intra vitam* and is one of the few nonfatal cases of infantile toxoplasmosis. Moreover, 1 case of hereditary chorioretinitis, 1 case of chorioretinitis in chronic encephalitis and 1 case of encephalitis with chorioretinitis and intracerebral calcifications are described.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

91:589-664 (March 15) 1947

*Chronic Form of Brucellosis. J. C. Gerrits.—p. 600.
Therapeutic Results in Treatment of Urinary Infections in Department for Internal Diseases. L. Loopuyt.—p. 611.
Neurosurgical Treatment of Cerebral Abscess. C. H. Lenshoek.—p. 616.

Chronic Brucellosis.—Gerrits points out that chronic brucellosis closely resembles the onset of pulmonary tuberculosis. He presents histories of 2 patients with recurring hemoptyses. Pulmonary tuberculosis had been suspected, but the condition was diagnosed as chronic brucellosis on the basis of the clinical picture. In the first patient this diagnosis was verified by a positive skin test and in the second by a positive agglutination reaction of 1:100 and a positive skin test. The author investigated the possibility of brucellosis in patients in whom tuberculosis had been suspected because of fatigue, night sweats, subfebrile temperature and hemoptysis but in whom tuberculosis could be excluded. In 3 of 66 patients sarcoma, carcinoma and syphilis were detected, but in the others the diagnosis remained obscure. A skin test for brucellosis was positive in 33 of the remaining 63 patients. In some of these patients brucellosis can be diagnosed on the basis of the clinical symptoms. Of 40 patients in whom brucellosis was not suspected, only 3 gave positive skin tests.

91:729-788 (March 29) 1947

*Pulse and Respiratory Frequency in Malaria. E. van der Kuyp.—p. 735.
Penicillin Intoxication? J. J. H. M. Klessens.—p. 740.
Experimental Psychologic Investigations on Astereognosis: Physiology and Pathology of Stereognosis. J. J. G. Prick and P. J. A. Calon. —p. 745.

Pulse and Respiratory Frequency in Malaria.—Van der Kuyp reviews observations on the temperature, pulse rate and respiration of 130 patients with quartan malaria and of patients with tertian or subtertian malaria observed at a hospital in Paramaribo. Pulse and respiration curves showed the quartan, tertian or subtertian type more constantly and more clearly than did the temperature curves. The three curves often supplemented one another in such a way as to make the type of fever clear. The pulse curve sometimes showed the existence of a duplex or triplex quartan infection. During treatment the temperature was the first to become normal. In patients in whom the pulse and respiration did not become normal, a relapse followed. The characteristic course suggests that schizogony on a small scale is responsible for the changes in pulse rate and respiration, because the quantity of liberated toxins is too small to influence the temperature but large enough to influence the pulse and respiratory rate, these being more sensitive to these toxins.

Acta Pædiatrica, Stockholm

33:1-114 (Oct. 31) 1945

Prognosis for Bronchial Asthma Arisen in Infancy, After Nonspecific Treatment Hitherto Applied. E. W. Flensborg.—p. 4.
*Genesis of Erythroblastosis Fetalis. S. Ranström.—p. 25.
*Acute Benign Mononuclear Meningitis. E. Jacobsson.—p. 37.
Allergic Purpura with Hypoprothrombinemia. A. Njå.—p. 54.
Familial Congenital Hypertrichosis Totalis (Trichostasis). T. A. E. Janssen and Cornelia de Lange.—p. 69.
Clinical Appearance and Diagnosis of Colon Polypus. J. Lewis-Jonsson. —p. 79.
*Gastroduodenal Ulcer in Childhood. E. Wamberg.—p. 86.
Genu Varum in Children: Typical Roentgen Picture. A. Renander. —p. 98.
Knobby Formations in Cranial Bones in Severe Anemias During Childhood. V. Liakka.—p. 104.

Genesis of Erythroblastosis Fetalis.—Ranström says that since the formulation of the Rh isoimmunization theory of erythroblastosis fetalis, it has usually been referred to as hemolytic disease of the newborn. This theory of Rh isoimmunization raises a number of questions for which there is no satisfactory answer. It remains to be explained (1) why signs of Rh isoimmunization are absent in a number of cases of erythroblastosis, (2) why Rh isoimmunization can be shown to

be present without erythroblastosis and (3) why the clinical course of the disease is not satisfactorily explained by the Rh isoimmunization theory. The author regards a placental lesion as an indispensable prerequisite of an isoimmunization and points out that before the Rh theory was advanced a number of observers had noted changes in certain of the endocrine organs. He reports 2 cases which in addition to adrenal hyperplasia and in 1 case of insular hyperplasia in the pancreas showed pituitary changes hitherto not described. These pituitary changes consisted of an increase in basophil cells. The theory of Rh isoimmunization does not explain the disturbances in the endocrine organs. The author advances the theory that the primary element in the disease is an endocrine dysfunction in the mother, which induces a placental change and changes in the endocrine organs of the fetus (perhaps mainly via its hypophysis), with resulting dysfunctions according to the character of these changes. The data in the literature point to an abnormally high production of estrone or gonadotropic hormone. In some cases, especially in hydrops fetalis, the endocrine disturbances are thought to determine the issue. When the appropriate serologic conditions are present, the placental lesion paves the way for an Rh isoimmunization, which in turn is considered to influence the appearance of icterus gravis and anemia gravis neonatorum. The author outlines an endocrine therapy for erythroblastosis fetalis on the basis of this theory.

Acute Benign Mononuclear Meningitis.—Jacobsson reviews observations on 25 cases of acute benign mononuclear meningitis treated at the Gothenburg Children's Hospital since 1922 and presents the case histories for those reported during the past fifteen years. The relations of the disease to poliomyelitis and encephalitis are discussed, it being claimed that no absolutely certain connection between them has heretofore been demonstrated. As a rule the course of the disease is rapid and benign. There is no specific therapy, nor is it necessary.

Gastroduodenal Ulcer in Childhood.—Wamberg reports that a girl aged 9 years had had periodic vomiting for five years and that a girl aged 11 years had had attacks of vomiting for two years as well as periodic, partly tardy pain in the epigastric region and melena twice. Ewald's test meal and x-ray examination revealed a typical duodenal ulcer in both patients. Ulcerations of the stomach and duodenum are most frequently encountered in the first year of life and around puberty. In infancy the ulcers nearly always have appeared in conjunction with infections, intoxications, traumas, circulatory disorders, burns or cachexia. The ulcers occurring in children above 5 years of age in the vast majority of cases are based on gastroduodenitis. The disease may manifest itself by (*a*) a gastroduodenitis syndrome, just as in adults, (*b*) the sudden occurrence of complications (hemorrhage or perforation) and most often by (*c*) vague and atypical symptoms. The treatment should be conservative. Gastroduodenal ulcers in childhood presumably occur more frequently than is generally supposed. A more extensive use of x-rays in the examination of children with atypical dyspeptic symptoms would lead to a more frequent recognition of the condition.

33:211-390 (Dec. 30) 1946. Partial Index

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Pneumothorax, Mediastinal Emphysema and Cutaneous Emphysema Complicating Tracheotomy. D. Riis.—p. 321.
*Onset and Prognosis of Nephritis in Children. Å. Frisk and G. Klackenberg.—p. 349.
Acute Macrocytic Anemia in Newborn, with Special Reference to Relation to Erythroblastosis Fetalis. S. Heinild.—p. 373.
Paroxysmal Tachycardia in Infants. E. Mannheimer.—p. 383.

Acute Leukemia in Children.—According to Riis, textbooks still describe leukemias as following an acute course, with high fever, hemorrhagic diathesis, necroses in the larynx and oral cavity and having a rapid course. A number of monographs in recent years have shown that this description applies only to the terminal stages of the disease. At the Pediatric Department of the University Hospital of Oslo 24 cases of acute leukemia have been diagnosed between 1937 and 1944. The patients were admitted under various diagnoses such as leukemia, anemia, acute rheumatism, scurvy with anemia, adenitis of the neck, mongolism, bronchopneumonia or disease of the blood. Fatigue, lack of appetite, pallor and fever were the most frequent initial symptoms. Enlargement of cervical lymph nodes and anemia are likewise frequent. In three fourths of the cases the disease began during the first five years of life. The acute leukemia in children as a rule is a myeloblastic leukemia (in at least 21 of 24 cases). In 18 cases sternal puncture was done successfully and revealed up to 99.4 per cent of myeloblasts. The pathologic type of cell was demonstrated in blood-smear preparations of all cases. The number of white cells varied from 1,300 to 185,000.

Schönlein-Henoch's Purpura After Prophylactic Vaccination Against Diphtheria.—Jelke observed a typical case of Schönlein-Henoch's purpura which developed two days after administration of toxoid. In addition to cutaneous hemorrhages there developed edemas of the extremities, articular pains, colic, occult intestinal hemorrhage and hematuria. The cutaneous hemorrhages appeared in successive crops over a period of five months. Patch tests with toxoid were negative, but intracutaneous injection of 0.001 cc. of a toxoid dilution resulted in intestinal hemorrhage, which proved that diphtheria toxoid was the eliciting allergen.

Onset and Prognosis of Nephritis in Children.—Frisk and Klackenberg thought that follow-up examinations a sufficient number of years after the onset of nephritis would throw light on the prognosis. Their studies were made on 239 patients, of whom 96 per cent were still alive from ten to twenty years after the first hospital treatment. In addition to the ordinary tests, quantitative sediment counts by Addis's method were made. Ninety per cent of the patients with acute nephritis, most of whom had had a hemorrhagic onset, had recovered. Slightly more than 5 per cent had permanent renal damage, and somewhat less than 5 per cent were borderline cases, where further investigation would have been necessary for definite classification. An unnoticed insidious onset was combined with bad prognosis. This form of the disease, termed primary chronic, led to death within five years. This type of onset is not common, accounting for only 11 cases. The secondary chronic, on the other hand, influenced the general state of health surprisingly little over a long period. Acute convulsive uremia and the extent of the initial albumin did not influence the prognosis, nor did tonsillectomy.

Nordisk Medicin, Gothenburg

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Folic Acid. J. Waldenström.—p. 399.
Health of Copenhagen School Children During War Years. K. Bojlen.—p. 403.

Hospitalstidende

*Significance of Positive Takata Reaction: Examination of 5,825 Serums Sent In for Wassermann Test. T. Fløystrup.—p. 407.
Sulfonamide Anuria. B. Viking.—p. 410.

Norsk Magasin for Lægevidenskapen

Metastatic Myositis with Glandular Abscess (Streptococcic Metastasis): Two Atypical Cases. O. Solvaag.—p. 429.
*Takata Reaction in Extrahepatic Disorders. H. F. Lange.—p. 432.

Hygiea

Value of Cholangiography. O. Schuberth.—p. 441.
Surgical Treatment of Megacolon. I. R. Sandberg.—p. 445.

Significance of Positive Takata Reaction.—Fløystrup reports that the Takata reaction was positive in 100 of 5,825 serums. In 83 cases injury of the liver parenchyma was established, hepatitis in 71, cirrhosis of the liver in 8. In 7 of the remaining 17 cases there was congestion of the liver. Of the 35 cases with maximal precipitation 29 were due to chronic hepatitis and 3 to cirrhosis of the liver. Although many serums must have come from patients with cholelithiasis, no positive reactions were recorded in these.

Takata Reaction in Extrahepatic Disorders.—Lange says that of 123 cases with positive Takata reactions about three fifths appeared in extrahepatic disorders, especially in chronic, nonspecific pulmonary diseases. A positive Takata reaction in pulmonary disorders is an unfavorable prognostic sign.

Book Notices

Biochemistry of Cancer. By Jesse P. Greenstein, Head Biochemist, Chairman, Section on Biochemistry, National Cancer Institute, National Institute of Health, United States Public Health Service, Bethesda, Maryland. Cloth. Price, $7.80. Pp. 389, with 39 illustrations. Academic Press, Inc., 125 E. 23d St., New York 10, 1947.

This book, which comprises in good part the extensive investigations of the author and others at the National Cancer Institute, discusses biochemical studies in the field of cancer research which have yielded results of interest and significance. As stated by the author in the preface, a desire to show the reader the tremendous gaps that exist in our knowledge and to illustrate the possibilities of reciprocal enrichment in the fields of normal and abnormal cell physiology are brought home.

The material is presented in four general categories, on general phenomena and taxonomy of cancer, the induction of tumors including extrinsic and intrinsic factors, attempts at control of tumor induction and growth by nutritional, endocrinologic and chemotherapeutic means, and the properties of tumors, which is divided into a long chapter on the chemistry of tumors and another on the chemistry of the tumor bearing host.

The second chapter, on the general phenomena and taxonomy of cancer, deals briefly with some characteristics of tumors and their transmissibility, and on the development and use of inbred strains of mice for experimental cancer research. The discovery of the carcinogenic polycyclic hydrocarbons and their many synthesized derivatives is discussed in relation to their comparative carcinogenic action in different sites of the body of many strains of mice, in chapter 3. Also the interesting problem of their metabolism is discussed and the dosage of and response to them in various organs of mice of inbred strains under many experimental conditions is given. A brief summary of the large amount of information on the azo dyes and their derivatives which are widely used as carcinogenic agents is well summarized.

The role of intrinsic factors, or those possessing a biogenic origin, which deal largely with the influence of sex hormones on the development of cancer of the sex and accessory sex organs, is delineated in chapter 4. Also the effect of intrinsic influences on the development of lymphoid tumors and their effect on liver and kidney arginase is given. The chapter is completed with a summary of the role of milk factor in mammary carcinogenesis in mice and of the well known viral origin of fowls and of the Shope rabbit papilloma. The nutritional, endocrinologic and chemotherapeutic approaches for their effects on tumor induction and on tumor growth are concisely and well summarized in the next three sections.

Especially interesting to biochemists engaged in cancer research or enzymology, and probably the most important chapter in the book, is the one on the chemistry of tumors, a large portion of the investigations being those of the author. From a comparison of many enzymes studied in different normal tissues and their corresponding neoplastic growths, especially liver and hepatoma, the general conclusions—that tumors have qualitatively the same enzymes as normal tissues, that tumors tend to resemble one another chemically more than they do normal tissues, that a normal tissue in becoming cancerous may lose some of its specific functions altogether or contain them to a less degree than normal, and that tumors tend to converge to a common type of tissue—are of great value in a clarification of the conception of carcinogenesis.

The final chapter is on the effects produced in an animal as a consequence of the presence of a tumor; to those not aware of this phase of cancer research the results will be most enlightening. The effect of various tumors transplanted under the skin on lowering the hepatic and renal catalase, d-amino acid oxidase and arginase is quite dramatic. The possibility of using these changes or, better yet, of searching for possible subtle blood changes caused by transplanted and induced tumors for diagnostic purposes will be of obvious importance to all.

The size and format of the book are convenient and it appears to be relatively free of errors. It will serve as an excellent reference work for the student interested in cancer research, though in some phases it is not complete.

Concise Chemical and Technical Dictionary. Edited by H. Bennett, Technical Director, Glyco Products Co., Inc. Cloth. Price, $10. Pp. 1,055. Chemical Publishing Co., Inc., 26 Court St., Brooklyn 2, N. Y., 1947.

This type of book, if properly presented, is a useful addition to medical libraries. The student, teacher and physician today need information on a variety of chemical and technical subjects that have come about because of modern research. As soon as new ideas and substances are found to have practical value in medicine, the medical profession is introduced to new terms. The best sources of information frequently are not available and, if they are, contain detailed information that is not generally needed for the practitioner unless he has a special interest in the subject. The research worker, however, must refer to original publications. To meet such a need dictionaries can serve a useful purpose. However, they must be reasonably complete, accurate and informative to be useful for those interested in medicine. Bennett's Concise Chemical and Technical Dictionary cannot be regarded as satisfactory for this purpose, although it may have limited usefulness for those who want only casual information. Critics will not be satisfied with some of the definitions; the scope is not adequate and in a number of instances there is an unnecessary and perhaps confusing repetition; cross references are not always complete; there are various abbreviations that are not, so far as chemists are concerned, in common use, for example M. A. R., I. R., I. V. and I. W. It is disconcerting to look for a definition of bougie and be referred to "pencil" and not able to find a definition for pencil. It is rather irritating to find valuable space taken by definitions for words such as "tweed," which can hardly be classed as a word normally found in a chemical and technical dictionary unless the volume is of considerable size. Such a book as this could be much more useful if it were more complete and contained information that would reveal to the reader whether the name is protected or a common or generic name, what firm holds the name, more information on cosmetics, and whether the book is limited to the class of readers for which it is written; it is difficult to write a chemical and technical dictionary "for both professionals and laymen." About 50,000 definitions are claimed to be included in the volume, but they do not as stated "cover every field of scientific and technical development" except in a broad way. Pharmacists, physicians and those interested in cosmetics would like to see a better definition, for example, for night cream than "greasy type of cosmetic cream" and for penicillin "antibiotic substance found in the filtrate of a broth culture of Penicillium notatum." There might also be consistency in describing the uses of a compound; they could either be omitted or made more complete; for example, barbital is "used in medicine," while barbital soluble is "used as sedative, hypnotic." The author is to be commended for his attempt to undertake such a project; a book of this type is always needed. Unfortunately, however, as far as the medical profession is concerned, the results of the author's efforts cannot be as highly commended.

Pre-Frontal Leucotomy in 1,000 Cases. Great Britain, Board of Control (England and Wales). Paper. Price, 6d. Pp. 31. British Information Services, 30 Rockefeller Plaza, New York 20; His Majesty's Stationery Office, York House, Kingsway, London, W. C. 2, 1947.

Prefrontal leukotomy has been adopted on a fairly large scale in England as a method of treatment for mental disorders that have proved refractory to other methods. This is a report by the Board of Control covering results in 1,000 cases from forty-three different hospitals. Thirty-five per cent of the patients have been discharged, and 32 per cent are improved though still remaining in the hospital. The operative mortality is 3 per cent, mostly due to hemorrhage. Only 1 per cent of the patients are considered worse as the result of operation. These figures, gratifying as they are, hardly tell the whole story in terms of relief from intolerable preoccupations and misery on the part of the patients and of reduction in dangerous and disturbed behavior on the part of hospital inmates. Only the most difficult patients have been subjected to leukotomy.

In the small monograph the figures are broken down as regards age, sex, diagnosis, duration and so on. Results are better in older patients with shorter duration and in the affective group rather than the schizophrenic. Leukotomy (lobotomy in

this country) is particularly effective in relieving mental tension and depression, while hallucinations and delusions react less favorably. Relapses are of moderate frequency; epileptic fits and other complications are not very uncommon aftermaths; 242 patients are listed as "citizens" able to earn a living or keep house. While some brilliant results are reported in patients that have been institutionalized for a number of years, the striking feature of the report is the large number of successful results in those operated on within two years of the onset of the disease. Taking only the early cases, 50 per cent of the schizophrenic and 65 per cent of the manic-depressive group are discharged recovered if operated on within two years. The percentages fall to 10 or 15 in those operated on five years or more after onset. Even so, the improvement in hospital adjustment in these chronic cases runs around 40 per cent. "Remarkable improvement in behavior follows in a large percentage of cases who have had severe symptoms with poor prognosis and have failed to respond to other methods of treatment. Many are discharged from the hospital and others, while unfit to leave, become much more placid and easier to nurse. The question of whether or not these results are achieved at the cost of the loss of some finer mental qualities is not yet answered and further study is needed on this most important point."

This study, documented by several tables, answers many of the questions that are being asked concerning the surgical treatment of mental disorders. Other questions are propounded, but in view of the difficulties presented in gathering the material they can probably be answered better by individual investigators who follow their cases for long periods.

Chemotherapy Yesterday, To-Day, and To-Morrow. By Sir Alexander Fleming, M.B., F.R.C.S., F.R.C.P., Professor of Bacteriology, University of London, St. Mary's Hospital, London, W. 2. The Linacre Lecture Delivered at Cambridge on May 6, 1946. Paper. Price, 50 cents; 2s. Pp. 39, with 7 illustrations. Macmillan Co., 60 Fifth Ave., New York 11; Cambridge University Press, Bentley House, 200 Euston Rd., London, N. W. 1, 1946.

This is the Linacre Lecture of 1946 delivered at Cambridge University by the discoverer of penicillin. In discussing chemotherapy of the past, Fleming alludes to the observations of Pasteur, Lister, Ehrlich and himself. Fleming considers as his most important investigation work carried out in 1924 relating to the destructive action of antiseptics on the blood leukocytes. Leukocytes are essential for the phagocytosis and destruction of bacteria. Unfortunately, chemicals then available were not specific for bacteria alone but also destroyed the leukocytes. This work clearly defined the attributes of the ideal chemotherapeutic agent, one that would have a specific action against bacteria without altering the function of the host's tissue and blood cells. The sulfonamides constituted a definite step forward in pursuit of the ideal agent, but penicillin represents a highly potent antibacterial agent having surprisingly few toxic effects. Without any doubt, the earlier studies of Fleming alerted him for the potentialities of a fungus which appeared to destroy staphylococci on an agar plate. He concludes his lecture by appealing for the establishment in England of an institute of general microbiology in which the various disciplines of biology would be represented. This brief summary of chemotherapy should appeal to any one interested in the field.

The Nervous Child. By Hector Charles Cameron, M.A., M.D., F.R.C.P., Consulting Physician to The Children's Department, Guy's Hospital, London. Oxford Medical Publications. Fifth edition. Cloth. Price, $3. Pp. 252, with illustrations. Oxford University Press, 114 Fifth Ave., New York 11; Amen House, Warwick Sq., London, E. C. 4, 1946.

Since the first edition in 1919, many advances have been made in diagnosis and management of the so-called nervous child. The early chapters, on doctors, mothers and children, observations in the nursery, management and conduct, want of appetite, indigestion and want of sleep, are still as acceptable today as formerly, but there are several later chapters which are quite unscientific, outdated and in need of thorough revision. Formerly there were few good books in this field. There are now many which are more complete and are able to replace the old textbooks.

Essentials of Physiological Chemistry. By Arthur K. Anderson, Professor of Physiological Chemistry, The Pennsylvania State College, State College, Pennsylvania. Third edition. Cloth. Price, $3.50. Pp. 395, with 38 illustrations. John Wiley & Sons, Inc., 440 Fourth Ave., New York 16; Chapman & Hall, Ltd., 37-39 Essex St., Strand, London, W. C. 2, 1947.

This volume has been revised, and certain sections have been rewritten. The text contains materials essential to students for a short course in chemistry entering medicine, bacteriology, nutrition and allied sciences. A discussion of optical isomerism has been brought up to date, glyceric aldehyde rather than lactic acid being used as an example, carbohydrate metabolism has been further developed and the section on chemotherapy, which contains newer drugs and antibodies, has been expanded. The vitamins have been brought up to date, and a chapter on blood has been added. The volume will be a good addition to a collection of books on physiologic chemistry.

Obstetricia de urgencia: Sindromes hemorragicos. Por los doctores Diego Taylor Gorostiaga, profesor suplente de clínica obstétrica normal y patológica de la Facultad de ciencias médicas de la Universidad nacional de La Plata, y Roberto Enrique Lede, jefe del servico de obstetricia y ginecología del Hospital Petrona Villegas de Cordero. Prologo del Prof. Dr. Victorio Monteverde, profesor titular de clínica obstétrica normal y patológica de la Facultad de ciencias médicas de la Universidad nacional de La Plata. Paper. Pp. 419, with 103 illustrations. López & Etchegoyen S. R. L., Junin 863, Buenos Aires, 1946.

This book deals with obstetric emergencies. In the sixteen chapters the authors describe, illustrate and discuss the etiology, diagnosis and treatment of abortion, hydatid mole, ectopic pregnancy, placenta previa, abruptio placentae, acute torsion of the gravid uterus, rupture of the uterus, postpartum hemorrhage, bleeding elsewhere than from the uterus, inversion of the uterus and shock. An entire chapter consisting of fifty-five pages and twenty-seven illustrations is devoted to blood transfusion and the use of plasma. The text is written with simplicity, and the illustrations are numerous, clear, instructive and presented in an interesting fashion. The advice given by the authors is practical and apparently is based on an extensive experience. However, the Braxton Hicks version should be given up for the treatment of placenta previa because it is dangerous and intrauterine douches should have no place in the treatment of profuse uterine bleeding because, while the douche is being prepared and given, valuable blood is being lost. There are much quicker and more certain means of controlling uterine hemorrhage than intrauterine douches. The contents of this book may be found in all the standard textbooks of obstetrics, but it does bring together most of the emergency conditions that occur during pregnancy. For this reason it serves a useful purpose and is worthy of possession and study.

The 1946 Year Book of General Therapeutics, November, 1945-August, 1946. Edited by Oscar W. Bethea, Ph.M., M.D., F.A.C.P. Fabrikoid. Price, $3.75. Pp. 443, with 20 illustrations. Year Book Publishers, Inc., 304 S. Dearborn St., Chicago 4, 1946.

Those who are familiar with the Year Book of General Therapeutics will find in the 1946 volume essentially the usual approach for this series to therapeutic highlights for the year, in this case extending from November 1945 to August 1946. For others who have yet to make an acquaintance with the book, it may be pointed out that it consists essentially of an abstracting of current medical literature published during the year. Included are discussions of sulfonamides, sulfones, penicillin, streptomycin, amebicides, burns, antimalarials, antisyphilitics, antirheumatics, gout, serums and plasma, vaccines and other drugs such as folic acid, amino acids, hormones, vitamins, thiouracil, sedatives and those acting especially on the heart and blood vessels. If a reader wishes to have available a ready source of fairly comprehensive abstracts the Year Book will provide much help. If a reader wishes to be guided in his choice of therapy he may have to go elsewhere, as abstracts of various papers sometimes present conflicting views and, of course, occasionally present views not yet generally adopted or later disproved. There are many, however, who come to regard the Year Book series as useful additions to their libraries, and these persons will not be disappointed in the 1946 Year Book of General Therapeutics; it again provides what has come to be expected.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

AIR TRAVEL AND HYPERTENSION

To the Editor:—A woman aged 65 wishes to fly from Boston to California. She has arthritis, with a blood pressure of 238/120. Could or would such a thing as hitting an air pocket cause untoward effects and possible cerebral vascular episode? Disregarding the arthritis, would her blood pressure or hypertension rule out air transportation?

David Glunts, M.D., Roxbury, Mass.

ANSWER.—Transcontinental flying, which involves ascent to considerable altitude in crossing the Rocky Mountains, is inadvisable for a patient of 65 with hypertension of the degree mentioned in the query. While it is quite possible that the trip could be made without lasting injury, the probability of intracranial or cardiac difficulties is sufficient to advise against the undertaking. Histanoxia of tissues distal to the constricted arterioles is the chief source of injury to parenchymal tissues in hypertensive disease; the lowered oxygen tension of fairly high altitude flying may well be just enough to lower the oxygen supply to the brain or myocardium below critical levels. This hazard is intensified if the patient is also anemic or if her hypertensive disease has progressed to arteriosclerosis. Furthermore, the anxiety and excitement of such a trip can well lead to a transient but dangerous exacerbation of the hypertension. Travel in a railroad compartment is the safer method for this patient.

DIPHTHERIA TOXOID

To the Editor:—In the care of men and women of college age, how soon after initial immunization for diphtheria with alum precipitated diphtheria toxoid should a booster dose be given, continued at what intervals and what dose used?

F. F. Ebersole, M.D., Mount Vernon, Iowa.

ANSWER.—If negative Schick reactions have been obtained following immunization with alum precipitated toxoid, a booster dose would not ordinarily be given to men and women of college age. However, if diphtheria was prevalent in the community it would be well to perform Schick tests annually, and then a booster dose of alum precipitated diphtheria toxoid might be given to those who had positive Schick reactions. Under such circumstances, 1 cc. of alum precipitated toxoid may be injected subcutaneously. Nevertheless, it has been found that as little as 0.1 cc. of plain diphtheria toxoid intracutaneously is sometimes sufficient to bring about a change from a positive to a negative Schick reaction.

The duration for diphtheria immunity is greatly influenced by the number of injections of toxoid. When three doses are administered at four week intervals, immunity persists in more than 90 per cent of the cases for a period in excess of four years.

TIDAL WATER AND POLIOMYELITIS VIRUS

To the Editor:—I should be grateful for information as to any recent publication concerning water-borne diseases. What I have in mind is the relationship of various infections to polluted public waters, such as our Narragansett Bay, parts of which have recently been closed to shellfishing, bathing and other uses because of increasing contamination from untreated and imperfectly treated sewage. Contamination of the water supply for domestic use is not under consideration. Has any work been done that would indicate that salt or tidal water can spread the virus of poliomyelitis?

Paul C. Cook, M.D., Providence, R. I.

ANSWER.—The broad question of water contamination is amply covered in most textbooks on hygiene or water purification.

The second more specific question is "Can salt or tidal water spread the virus of poliomyelitis?" There has been much effort made to implicate water as a factor in the spread of this disease. Kling in Sweden claims to have recovered the virus from water. These claims have not been substantiated. Water obtained in streams or bays into which raw sewage is emptied has been found to be contaminated (Detroit and Charleston) but not for long, and the virus is soon diluted to a point where it must be of little epidemiologic importance. Swimming pools and drinking water have been investigated many times with fruitless results.

It has not been proved that water carries virus, and even though water did carry virus it would be of little epidemiologic importance.

DERMATOLOGY AS A SPECIALTY—FIRST DERMATOLOGIC AUTHORITY

To the Editor:—Will you kindly inform me as to when dermatology first became recognized as a separate specialty and name the first man who became a recognized authority on dermatology?

Wallace E. Hopkins, M.D., Dallastown, Pa.

ANSWER.—The first question as to when dermatology first became recognized as a separate specialty cannot be answered in one or two words. The recognition of the specialty was a gradual process lasting years and varied in different countries. For instance, the founder of French dermatology, as stated in Pusey's "History of Dermatology," was Antoine C. Lorry (1726-1783). It is generally accepted that the founder of British dermatology was Robert Willan (1757-1812). Dermatology was certainly recognized as a specialty in the United States in 1876, when the American Dermatological Association was founded. However, the first group of dermatologists to organize was the New York Dermatological Society in 1869, largely by men who had had their special training abroad.

To name the first man who became a recognized authority on dermatology is even more difficult. The honor should probably go to Jean Astruc (1684-1766), who was a brilliant man, a physician to the king in Paris, and a teacher of Lorry.

ANTHRAX IN ELEPHANTS

To the Editor:—Because of a fatal case of anthrax that has come to my attention may I ask whether there have been any human cases of anthrax traced to infection in elephants? Specifically, have human cases of anthrax been traced to work (such as cutting or carving) with elephant tusks imported from Africa?

Roy M. Seideman, M.D., West Hartford, Conn.

ANSWER.—Reference to human cases of anthrax traced to elephants cannot be found. No cases of human anthrax are known to have occurred in Africa as a result of working with elephants or from handling elephant tusks.

G. H. Evans reports anthrax as relatively common in elephants in India.

African elephants in the Belgian Congo are considered little subject to Bacillus anthracis infection. "Symptomatic anthrax" caused by Clostridium chauvoei does occur in hippopotamuses in the Belgian Congo. Considering the wide range of animals susceptible to anthrax, its prevalence in certain areas and the ability of anthrax spores to remain infective for long periods of time, it is not beyond the realm of possibility for anthrax to result from handling elephant tusks. Tusks are invariably grooved, pitted and cracked and could conceivably harbor anthrax spores.

SPASM OF ACCOMMODATION

To the Editor:—What should be done for patients with spasm of accommodation? In a woman retinoscopy reveals plus 150 sphere each eye with working distance deducted. All that she will accept is a plus 50 sphere right eye and plus 25 sphere left eye. She already has been fitted with six pairs of glasses by six different doctors. These lenses range from plus 2 spheres down to plus 25 sphere each eye.

Donald Brink, M.D., Hutchinson, Minn.

ANSWER.—On the basis of the information given in the query, it is not possible to establish a diagnosis of spasm of accommodation, which is an exceedingly uncommon condition. A certain degree of sustained accommodation is frequently seen in young hypermetropic persons and may be considered a physiologic adaptation in the interests of clear vision.

Excessive contraction of the ciliary muscle causes a relative myopia, increased actual myopia or increased latent hyperopia. It may be caused by miotics, drugs such as arsenic and the sulfonamides, ocular injuries and conditions causing irritation of the ciliary body. Most authorities indict excessive near work, defective illumination, uncorrected refractive errors, general debility and unstable disposition. Diagnosis is based on the observations of loss of accommodative amplitude, nearing of the far point and disappearance of the refractive abnormality with cycloplegia.

Treatment should be directed toward determination of the refractive error with full cycloplegia, using atropine if necessary, and the prescription of the full correction as found with the ciliary muscle paralyzed. In hypermetropic patients about 1 diopter is deducted to allow for the normal physiologic tone of the ciliary muscle. Many patients will require instillation of homatropine solution for a week or two after glasses are prescribed to allow recovery of the ciliary muscle from its condition of irritability. Near work should be forbidden for a time, its amount should be curtailed thereafter, and the general health should receive attention.

TREATMENT OF CONCURRENT CARDIOVASCULAR AND NEUROSYPHILIS

To the Editor:—**A man aged 51 was referred to a neuropsychiatric clinic because of convulsions and transient episodes of confusion. A diagnosis of dementia paralytica was established on the basis of typical spinal fluid findings. In addition, the patient had cardiovascular findings strongly suggestive of aortic insufficiency and syphilitic aortitis. There was no evidence of decompensation. The blood pressure was 142/20 in the left arm (in the sitting position) and 124/26 in the right arm. What treatment is indicated? Is penicillin contraindicated because of the cardiovascular involvement? Is the use of the hypertherm or of malaria contraindicated?**

Fred P. Pokrass, M.D., Norristown, Pa.

ANSWER.—Treatment of active cardiovascular syphilis with a rapidly spirillicidal drug such as penicillin or neoarsphenamine or a phenarsine derivative will frequently cause sufficient cardiac damage from therapeutic shock or paradox to result in cardiac decompensation or even, though less frequently, in sudden death. The danger of using penicillin in the presence of active cardiovascular syphilis was reported by Dolkart and Schwemlein in THE JOURNAL, October 13, 1945.

Although reports of this sort are not as yet frequent in the literature, probably because of the recent introduction of penicillin in the treatment of syphilis, similar cases, reports of which have not been published, have occurred.

When confronted with concurrent cardiovascular syphilis and neurosyphilis, one should make a careful appraisal of the relative seriousness of the two complications in the individual case, together with a study of previous therapy for syphilis if any. If there has been several months of antisyphilitic therapy recently, or if there is reason to think that the cardiovascular involvement is at present inactive, there would be less danger from the use of a trivalent arsenical or from penicillin. If there has been no preparatory treatment, the risk from the immediate introduction of these drugs would be great.

In general, life expectancy from severe cardiovascular syphilis with aortic regurgitation is less than from central nervous system involvement. An effort must be made, therefore, to find a treatment for the nervous system which will not injure the heart and great vessels but will rather assist in its arrest. Tryparsamide in dosage of 2 to 3 Gm. once a week intravenously will answer the purpose of treating the central nervous system without injuring the heart. A pentavalent arsenical such as tryparsamide has little effect on the cardiovascular system. Its use could be accomplished with courses of bismuth subsalicylate intramuscularly for the cardiac involvement given once a week for eight to ten weeks with a rest period of six to eight weeks between courses. It would be best to start with subtherapeutic doses of the bismuth preparation, perhaps 0.5 to 1 cc. (30 to 60 mg. of bismuth metal) once a week for the first six to eight weeks before increasing to a full weekly dose of 2 cc. (120 to 130 mg. of bismuth metal). After four to six months of such treatment, if it is well tolerated, the cardiovascular process should be rendered less active and it might be possible, after consultation, to consider the cautious use of penicillin at that time for the central nervous system involvement if the progress with tryparsamide has not been satisfactory. Involvement of the optic nerve is, of course, a contraindication to the use of tryparsamide, but the risk here also would have to be weighed in the light of the other activity described.

There is no absolute contraindication to the use of fever therapy in a case such as the one described in the query. In fact, patients with minor degrees of cardiovascular involvement and even with aneurysm tolerate fever therapy well if they are not decompensated. Such a decision depends on individual appraisal of the patient, however, and in general it would be safer to employ tryparsamide rather than malaria or hyperthermy in treating a patient with severe aortic regurgitation.

FLEXIBLE KONE DIAPHRAGM

To the Editor:—**What is the general opinion among competent gynecologists as to the use of rubber cervical diaphragms which are known as "flexible kone diaphragm."**

R. P. Fruehauf, M.D., Superior, Wis.

ANSWER.—As far as is known, no competent gynecologist or any other physician has published a paper on the use of the "flexible kone diaphragm." No physician's name is mentioned in the advertising circulars concerning the diaphragms, so most likely there have not been any scientific publications about this instrument. The points of merit claimed by the manufacturers for the flexible kone diaphragm are that (1) it is easy to apply, (2) it is easy to remove, (3) it is scientifically correct, (4) it is nonirritating, (5) it is comfortable while in use, (6) it is noninjurious, (7 there are no troublesome after-effects and (8) it may be kept in stock for three years without loss of resiliency. The stem of the flexible kone diaphragm consists of a flexible chromium wire coil spring covered with soft para rubber. A soft pliable rubber cap is attached at the lower end of the stem and soft rubber oval cones are fastened to the stem.

The manufacturers of the flexible kone diaphragm also make and sell the flexible kone pessary, which "consists of a framework of especially made pliable, rustless chromium wire of small diameter covered with a coating of pure soft rubber, approximately 1/32 of an inch thick. At one end of the pessary are two prongs and at the other end is a soft rubber button about the size of a nickel. On the stem (1¼ inches in length) are three soft rubber cones so placed as to fit the fusiform canal of the cervix. . . ."

Reports in the literature have pointed out the dangers attending the use of intrauterine wishbone pessaries. These are foreign bodies which often lead to infection of the uterine cavity, tubes and peritoneum. There is also risk of perforation of the uterus. In some cases the prongs become embedded in the uterine wall. Extrauterine pregnancy as well as intrauterine gestation has occurred in the presence of a stem pessary.

While the 1/32 inch rubber covering of the chromium wire may add some protection, the insertion of the instrument within the cervix or in the cervical canal and uterine cavity places it in the category of objectionable foreign bodies capable of doing harm.

J. P. Greenhill (1939 Year Book of Obstetrics and Gynecology, p. 411) says that "all gynecologists are agreed on the harmfulness of paraphernalia which is placed in the cervical canal or uterine cavity for purposes of preventing conception. Not only may these devices be harmful and occasionally fatal but they are by no means certain in preventing conception. Therefore, because of their ineffectiveness and their great danger and because there are many far simpler and safer procedures, mechanical devices should never be inserted into the cervical canal or the uterine cavity for the purpose of preventing conception."

CHOICE OF ESTROGENIC COMPOUND FOR MANAGEMENT OF MENOPAUSE

To the Editor:—**A woman aged 53, four years past the menopause, is able to control hot flashes and palpitations by diethylstilbestrol 0.5 mg. every other night. In two weeks' time, however, "spotting" begins and lasts until the diethylstilbestrol is discontinued. Increased dose results in increased flow. Physical and gynecologic examination is otherwise negative. Is there any other estrogen, preferably oral, that does not have this effect? Is it advisable to do a diagnostic curettage in this case? If so, how soon after discontinuing diethylstilbestrol should this be done? Is it safe to continue this medication despite bleeding?**

M.D., California.

ANSWER.—There is a rather large number of compounds which possess estrogenic effect and may give relief to women who are suffering from the menopausal syndrome. These compounds are not necessarily related chemically but they still have this therapeutic effect in common. Nevertheless there are minor qualitative variations in their physiologic effects on tissues. For example, estradiol will enhance the effect of progesterone on the endometrium under certain conditions, while diethylstilbestrol may actually inhibit the action of progesterone. There seems to be a definitely higher incidence of estrogenic bleeding in the menopausal patient on diethylstilbestrol than with most other estrogens. The production of hyperplasia of the endometrium seems also to be more easily accomplished with diethylstilbestrol than with some of the other compounds. The exact reasons for this are not well known, but it is acknowledged that diethylstilbestrol has a somewhat different metabolic fate than the natural estrogens such as estrone and estradiol. It is therefore suggested that to avoid uterine bleeding other oral estrogens be used such as estrone sulfate, benzestrol or ethinyl estradiol. It is also suggested that some of the injectable diethylstilbestrol compounds such as the propionate or the palmitate may be found effective in controlling the patient's symptoms and still not produce the unwanted uterine bleeding. This may appear confusing, but it should be remembered that these compounds are absorbed slowly from the site of injection and the effective amount in the blood stream at any one time is probably small and will not produce bleeding because this phenomenon seems related to the rapid absorption of estrogens, with the development of sharp peaks in estrogen blood levels.

It is not wise to continue diethylstilbestrol by mouth while the patient is bleeding from the uterus, since hemorrhages may actually occur under these conditions. Four to five weeks after discontinuing diethylstilbestrol should be sufficient time before the new oral estrogens are attempted or the injectable estrogens administered. There appears to be little need of a curettage if the physician is convinced that the cause of the bleeding is due to estrogen therapy.

TURPENTINE FUMES AND REACTIVATION OF TUBERCULOSIS OF KIDNEY

To the Editor:—A man aged 45, a painter in previous good health, while working in a closed room using oil and turpentine paints became violently ill from the fumes, resulting in severe renal irritation, including classic symptoms of nephritis and cystitis. He never recovered his health and subsequently, within a period of weeks, was found to have a flare-up of an old healed renal tuberculosis. Roentgenograms of the lungs did not reveal any pulmonary tuberculosis. He underwent a nephrectomy, and subsequent repeated cultures of the urine did not show further tuberculosis either of the bladder or of the surviving kidney. He still has frequency of urination and some suprapubic discomfort, but cystoscopy does not show gross pathologic changes. His claim for compensation was turned down and he is now appealing the case. Granted that tuberculosis is caused by the Mycobacterium of tuberculosis, would acute turpentine (and other paint solvents) nephritis cause the exacerbation of the latent renal tuberculosis? Would cystitis caused by turpentine explain the continued bladder symptoms, since further genitourinary tuberculosis has been ruled out? M.D., Arizona.

Answer.—There is no doubt that turpentine absorbed in sufficient amounts can cause severe acute renal damage with parenchymal necrosis and hemorrhage. Irritation of the lower urinary tract, including the bladder, is also an accepted fact. The question of the relation between the acute injury of the kidney by turpentine and the flare-up of an old healed renal tuberculosis cannot be answered without more detailed knowledge of the type of renal tuberculosis, of the evidence for its healed state and of the appearance of the gross and microscopic changes in the removed kidney. As in the case of the relation of trauma to tuberculosis, two mechanisms for reactivation of the infection are possible: (1) the local extension of tuberculosis from a latent focus due to direct injury of surrounding tissues and the opening up of pathways for the spread of tubercle bacilli; (2) the flare-up of local infection due to lowering of the patient's general resistance.

It is therefore debatable whether severe turpentine injury of the kidney might have damaged the region surrounding the old tuberculous focus enough to initiate a rapid local spread, or whether the deleterious general effects of turpentine poisoning lowered the patient's resistance and thereby led to the reactivation of the renal tuberculosis. The time interval between the onset of symptoms due to turpentine and the flare-up of the renal tuberculosis would seem to be appropriate for a causal connection between them, assuming that the patient was really free from symptoms of active renal tuberculosis previously.

The continuing bladder symptoms could scarcely be attributed to the original irritation by turpentine in view of the absence of objective findings and the lapse of time. Some other cause, including the possibility of a functional basis, should be considered.

The possibility that other paint solvents were responsible for the renal irritation is rather remote. At any rate, turpentine by itself could easily account for the symptoms.

SEMINAL FLUID EXAMINATION; EFFECT OF ABSENT TESTIS OR VARICOCELE

To the Editor:—What is the accepted "rough" method of making a sperm count which can be used in office practice? What are the limits of normal and the accepted criteria for determining whether a male is fertile? Does loss of one testis lower the sperm count? Would a symptomatic varicocele lower the sperm count? M.D., Pennsylvania.

Answer.—There is no "rough" method of making an examination of the seminal fluid to determine whether or not a man is fertile. The accepted procedure for seminal fluid analysis is as follows: After the man has abstained from sexual intercourse for five to six days, the seminal fluid is collected in a small beaker by having the patient ejaculate the semen directly into the container by artificial means. Condom specimens are not accurate. After liquefaction of the seminal fluid has occurred, which in normal specimens is from ten to fifteen minutes, the amount of semen is accurately measured in cubic centimeters. A small drop is then placed on a glass slide and sealed with a cover slide. The motility and percentage of active spermatozoa are estimated by using the high dry objective. Pus cells and bacteria are searched for. In normal specimens about 80 to 90 per cent of the spermatozoa show active motility. The longevity of the spermatozoa can be determined by drawing a small amount into a fine glass tube, sealing both ends, placing it in a refrigerator and making routine observations. Under ordinary ice box temperature, spermatozoa will be active after twenty-four to forty-eight hours.

To count the spermatozoa a sample is drawn up to the 0.5 mark in the white cell counting pipet while it is held nearly horizontal. A solution containing 1 per cent solution of formaldehyde and 5 per cent sodium carbonate in water is then drawn up to the 11 mark. The dilution is 1:20. A drop of the diluted seminal fluid is allowed to flow under the cover glass of the counting chamber. After allowing about five minutes for settling, one counts all the spermatozoa in the four large squares (each one 1 mm. square and 1 mm. deep), using the high dry objective. The total number of spermatozoa counted is then multiplied by 20 (for dilution) divided by 4 (number of cells counted) multiplied by 10 (for cubic millimeters, since the depth of the counting chamber is 0.1 mm.) and by 1,000 for cubic centimeters. The result obtained is the number of spermatozoa in 1 cubic centimeter. To obtain the total number of spermatozoa ejaculated, the number in 1 cc. of seminal fluid is multiplied by the volume of seminal fluid. Normal specimens contain around 100,000,000 spermatozoa per cubic centimeter, or a total number of 250,000,000. A fertile man should have at least a total of 100,000,000 .spermatozoa. However, there are many other factors besides the number of spermatozoa that enter into a normal seminal fluid. The morphology of the spermatozoa should be studied; for this there are many different staining methods which can be obtained in any of the standard textbooks on fertility.

The absence of one testis, if the other one is normal, will not appreciably influence the total number of spermatozoa. Varicocele would lower the spermatozoa count if the testis on the side of the varicocele is not normal and the other testis is abnormal. However, if the other testis is normal and the seminal fluid analysis is within normal limits, the patient is potentially fertile.

MAGNESIUM SULFATE FOR VERTIGO AND MÉNIÈRE'S DISEASE

To the Editor:—A patient with severe vertigo has been treated and reports cure from the intravenous injection of 50 per cent solution magnesium sulfate 2 cc. three times weekly and ammonium chloride by mouth. She stated that during injection she felt as if she was burning up, but this passed off in a few minutes and she felt perfectly well, having no more dizziness for some months. Is the intravenous injection of magnesium sulfate in this manner beneficial and safe? M.D., Indiana.

Answer.—Magnesium sulfate has not been used extensively in the treatment of vertigo. Its dominant action physiologically consists of depression and inhibition. Given parenterally it has a diuretic effect. The magnesium ion is toxic when high concentrations accumulate in the extracellular fluid. If the kidneys are functioning in a normal manner magnesium is so rapidly excreted that change in blood level of the ion cannot be detected. The drug should not be employed in subjects who have renal insufficiency.

Schick reports its use in the treatment of 18 subjects with Ménière's syndrome who apparently had been refractory to other forms of treatment. The treatment consisted in intravenous injections of 5 cc. of 50 per cent magnesium sulfate solution administered two or three times weekly. The number of injections ranged from ten to twenty, depending on the response and the severity of the symptom complex in each individual. Each injection was administered at an extremely slow rate with the patient in the recumbent position. By carefully observing these precautions the patients did not observe any ill effects from the drug.

Of the 18 patients treated, 7 did not experience attacks during a control period of fourteen months, 7 noted definite improvement and 4 noted no change.

AMPHETAMINE COMPOUND AND WEIGHT REDUCTION

To the Editor:—What is the value of dexedrine sulfate, recommended to curb the appetite in cases of obesity? Is it safe? I realize that dietary restriction is the scientific approach to this problem, but to keep these patients on a diet is more theoretical than practical.

Nathan J. Frenn, M.D., Bark River, Mich.

Answer.—Amphetamine (benzedrine) sulfate and its dextrorotatory component dexedrine sulfate have been widely advocated in the treatment of obesity, since they curb the patient's appetite and maintain his feeling of well-being despite a low caloric intake. However, as soon as the drugs are withdrawn the appetite returns in full and, unless a new food pattern has been established, the patient generally is unwilling to adhere to his former diet. There is also danger of habit formation, especially since the type of patient who is unable to restrain his appetite by his own will power will be one prone to habituation. Disagreeable side effects of a minor nature include insomnia, anxiety and palpitation.